The Industrial Disease Standards Panel is a Schedule 1 Agency of the Government of Ontario attached to the Ministry of Labour. The function of the Panel, as defined in Section 86p of the Workers' Compensation Act of Ontario, is:

(a) to investigate possible industrial diseases;

(b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;

(c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases; and

(d) to advise on eligibility rules regarding compensation for claims respecting industrial diseases.

The Panel is required by statute to report its findings to the Workers' Compensation Board of Ontario.

Additional copies of this publication are available by writing:

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SECOND REPORT TO THE WORKERS' COMPENSATION BOARD ON CERTAIN ISSUES ARISING FROM THE REPORT OF THE ROYAL COMMISSION ON ASBESTOS

Industrial Disease Standards Panel
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SECOND REPORT TO THE WORKERS' COMPENSATION BOARD

ON

CERTAIN ISSUES ARISING FROM THE REPORT OF

THE ROYAL COMMISSION ON ASBESTOS

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INDUSTRIAL DISEASE STANDARDS PANEL

IDSP REPORT NO. 7

APRIL, 1990

Ontario Ministry of Labour
Ministère du Travail de l'Ontario

Industrial Disease Standards Panel
Comité des normes en matière de maladies professionnelles

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April 24, 1990

Dr. R.G. Elgie
Chairman
Workers' Compensation Board
2 Bloor Street East, 20th Floor
Toronto, Ontario
M4W 3C3

Dear Dr. Elgie:

In accordance with Section 86p(10) of the Workers' Compensation Act, I am pleased to convey herewith IDSP Report No. 7 which addresses the issue of asbestos-related cancers.

Yours sincerely,

J. Stefan Dupré
Chairman

JSD:jgh

Ontario Ministry of Labour
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April 18, 1990

MEMORANDUM TO: **WORKERS' COMPENSATION BOARD**

FROM: **INDUSTRIAL DISEASE STANDARDS PANEL**

RE: **SECOND REPORT ON CERTAIN ISSUES ARISING FROM THE REPORT OF THE ROYAL COMMISSION ON ASBESTOS**

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1. BACKGROUND AND ISSUES

1.1 On September 21, 1988, the Industrial Disease Standards Panel (the 'Panel') issued its **FIRST REPORT TO THE WORKERS' COMPENSATION BOARD ON CERTAIN ISSUES ARISING FROM THE REPORT OF THE ROYAL COMMISSION ON ASBESTOS** (IDSP Report No. 4). In that Report, the Panel expressed its intention to undertake a comprehensive examination of all the recommendations of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario (RCA) which remained outstanding and fell within the Panel's statutory mandate. In addressing this task, the Panel placed the numerous RCA recommendations which were involved into four categories. In its **FIRST REPORT**, the Panel addressed one of these four categories, namely the set of issues first raised by the RCA concerning which it was satisfied that it had "little or nothing to add to the voluminous evidentiary base compiled by the RCA in the course of its research program and public hearings". The **FIRST REPORT** addressed RCA recommendations on the scheduling of asbestosis and mesothelioma; survivors' benefits for workers previously receiving partial disability awards for asbestosis; notification procedures for claimants and survivors; compensation for psychological impairment for victims of asbestosis; and organizational and structural reforms pertinent to the processing of asbestosis claims.

1.2 The remaining three categories of activity identified by the Panel encompass the following:

A. **ASBESTOS-RELATED ISSUES**

In this **SECOND REPORT TO THE WCB ON CERTAIN ISSUES ARISING FROM THE REPORT OF THE ROYAL COMMISSION ON ASBESTOS**, the Panel is making recommendations on cancers related to occupational asbestos exposure pursuant to RCA Recommendation 12.12 (RCA Final Report, 1984; Vol. 3, Ch.12, p.714) and to a Board request (R.C.Elgie, March 5, 1987). The Panel is continuing to examine the compensation of survivors of asbestos workers with a prior diagnosis of asbestos-related health effects (viz. asbestosis) who die from cardiovascular disease (viz. cor pulmonale).

B. **REVIEW OF OCCUPATIONAL DISEASES AND MECHANISMS FOR THEIR COMPENSATION**

The Panel has examined the statutory underpinnings in the Ontario Workers' Compensation Act (the 'Act') for the adjudication of compensation claims for industrial disease including schedules, eligibility rules and the benefit of doubt provision (Section 3(4)). This examination has included the commissioning of a discussion document on the subject by Professor Terence Ison of Osgoode Hall Law School ('Compensation for Industrial Disease Under the Workers' Compensation Act of Ontario' by T.G. Ison. IDSP Occasional Paper, September, 1989). Also, the Panel has convened a Round Table Meeting to promote informed discussion on adjudicatory issues in industrial disease. This discussion, whose participants included scientists, lawyers, physicians and representatives from the interested community, will be summarised in the Panel's 1989 Annual Report. With the benefit of this discussion, the Panel is now in a better position to make informed recommendations on all industrial diseases including asbestos-related cancers, the subject of this **SECOND REPORT**. Beyond these cancers, the Panel will launch a review for compensation purposes of all occupational diseases. This review will include, in particular, those diseases generally identified by the RCA as chronic and life shortening and which should entail compensation for psychological as well as physical impairment.

C. **ACTIVITIES TO PROMOTE THE ETHICAL DEVELOPMENT OF OCCUPATIONAL EXPOSURE AND DISEASE DATA:**

These activities include consideration of the following:

i) **Exposure Registries** (e.g. for PCB-exposed workers in IDSP Report No. 2):

ii) **A Disease Cluster Registry** (1987-88 Annual Report);

iii) **Principles for the Effective and Ethical Investigation and Reporting of Disease Clusters**. In its 1987-88 Annual Report, the Panel solicited advice on this subject and a digest of the responses with commentary will appear in the 1989 Annual Report; and

iv) An **Occupational Cancer Data Base**, options for which were first identified in a report commissioned by the Panel ('Occupation and Cancer in Ontario: Review of the Options for Establishing a Cancer-Occupation Data Base for Ontario' by L.D. Marrett and E. Weir, IDSP Occasional Paper, May 24, 1989).

v) **Monitoring Activities**: These include monitoring the effectiveness and ethics of outreach and health surveillance programs (e.g. for asbestos-exposed workers).

2. FINDINGS AND ELIGIBILITY RULES

2.1 INTRODUCTION

2.1.1 Panel staff prepared a review of the epidemiological literature relating occupational asbestos exposure to mortality or morbidity from certain malignant diseases (shown in Appendix A as 'Report on the Relationship Between Occupational Asbestos Exposure and Malignant Diseases', K.S.Yeung, July 5, 1989). The diseases included in this review were lung cancer, mesothelioma, cancer of the larynx, and cancers of the gastrointestinal or GI tract (specifically the esophagus, stomach, colon and rectum). As regards mesothelioma from occupational asbestos exposure, a recommendation to place this industrial disease in Schedule 4 of the Act was contained in Panel's First Asbestos Report. The Board responded by placing this disease with restrictions in both Schedules 3 and 4 of the Act (The Ontario Gazette, Oct. 21, 1989).

2.1.2 For the purposes of this Report, the staff review, whose text

comprises Appendix A, is relevant to the Panel's consideration of asbestos-related lung, laryngeal and GI cancers. The review concentrates especially on the published epidemiological literature which emerged following the issuance of the Report of the Royal Commission on Asbestos in 1984. The epidemiological evidence considered for each cancer site includes the evidence for work-relatedness: the effects of industrial processes; of age at first exposure; of intensity, duration and accumulated dose of exposure; and of disease latency.

2.2 LUNG CANCER

2.2.1 The post-RCA epidemiological literature has reinforced the causal relationship of this disease with occupational exposure to asbestos. The RCA report listed a number of cohort studies of asbestos workers in the following industries all showing excess lung cancer risk: mining and milling, friction materials, textiles, cement products, general manufacturing, gas mask manufacturing, insulation products, insulators and shipyard workers (Table 5.18, RCA Report, Vol.1, ch.5, pp.226-231). The post-RCA literature extends this list to include: general manufacturing; "any asbestos work" industries; electro-chemical, sheet metal, plumbing and pipefitting industries; and railroad work (App. A, Table 1, pp.66-71). As a consequence, the Panel makes the following finding:

FINDING 1: THE PANEL CONFIRMS THE EXISTENCE OF A PROBABLE CONNECTION BETWEEN CANCER OF THE TRACHEA, BRONCHUS AND LUNG AND OCCUPATIONAL EXPOSURE TO ASBESTOS.

2.2.2 The Panel notes that the observed excess of lung cancer risk does appear to vary with the type of industrial process in which asbestos is used. And it agrees with other RCA conclusions regarding industry processes. For instance, health risks for this cancer among insulation workers continue to appear to be greater than the risks for miners and millers and most other manufacturing workers.

2.2.3 The accepted view at the time of the RCA was that of the three principal fibre types, chrysotile was the least hazardous, crocidolite the most hazardous, while amosite occupied a position intermediate to the other two. As noted at the time however,

this view was challenged by those who believed that the evidence did not warrant any differentiation on the basis of fibre type (RCA Report, Vol.1, ch.5, pp.231-2). The Panel notes that this controversy continues with few changes.

2.2.4 To illustrate, the RCA cited a followup study of workers in a New Orleans asbestos cement manufacturing company as evidence in support of the proposition that crocidolite exposure led to a higher risk of lung cancer than did chrysotile exposure (Weill, 1979; Hughes, 1980). In these reports, this population had been followed up to 1974. The dose response relationship between the risk of lung cancer and estimated cumulative asbestos exposure provided suggestive but inconclusive evidence of a greater risk of lung cancer among workers exposed to a mixture of chrysotile and crocidolite asbestos than among those with exposure to chrysotile only. A further followup of this population through 1982 yielded additional supportive findings (Appendix A, p.45; Hughes, 1987). A comparison of lung cancer mortality among workers in each of the two plants in the study showed a higher risk for those exposed to both crocidolite and chrysotile than for those workers exposed mainly to chrysotile alone. Moreover, the author cited the consistency of this finding with a survey of 22 cohort studies of 30 populations by Doll and Peto (1985) which supports the proposition that amphiboles are more carcinogenic than chrysotile.

2.2.5 Nevertheless, the same author (Hughes, 1987) presents additional evidence showing similar dose response relations between risk of lung cancer and cumulative asbestos exposure for workers in one of the plants exposed only to chrysotile and those exposed to both chrysotile and crocidolite. Moreover, this observation of similar dose response relations by fibre type exposure is consistent with other findings in U.S. asbestos textile manufacturing plants (McDonald, 1983a; 1983b; 1985). In one of these plants, chrysotile alone was used; in the other, chrysotile, crocidolite and amosite were used.

2.2.6 The Hughes update of the New Orleans study aptly characterises the difficulties the Panel found in arriving at a clear and consistent picture of the relative contribution to lung cancer risk posed by different fibre types. Nevertheless, the conclusion (Doll and Peto, 1985, p.32-3) seems to hold that, all

other factors being equal, the hazard of lung cancer from chrysotile is less than that from crocidolite or amosite.

2.2.7 At the time of the RCA, the Panel notes that some one half of the successful compensation claims from asbestosis and mesothelioma arose from the Johns-Manville Scarborough plant. In this one Ontario plant, workers were variously exposed to chrysotile, crocidolite and amosite (RCA Report, Vol.1, ch.3, pp.117-9). Mixed exposures to different fibre types have not been an uncommon experience for asbestos workers. In fixed place industry, it can be difficult to confirm the mix of fibre exposures, and the difficulty is greatly compounded with respect to exposures arising in building construction, maintenance, renovation and demolition. Therefore, both because of the lack of complete clarity in differentiating risk by fibre type and the difficulty of ascertaining prevalence of historical worker exposures to mixed fibre types, the Panel finds it inappropriate to use this factor in formulating clear and consistent eligibility rules for the asbestos cancers.

2.2.8 With respect to age at first exposure, some recent studies which have separated the effect on lung cancer risk of age at first exposure from both duration of exposure and latency have found no effect (Appendix A, p.45; Hughes, 1987; Peto, 1985). This finding is consistent with a summary of evidence from North American insulation workers and British textile workers (Doll and Peto, 1985). In contrast, other studies have found a difference for age at first exposure: one study found an effect for women but not for men (Newhouse, 1985); another found a higher risk among men hired before age 25 than among those hired later (Alies-Patin, 1985). The RCA itself noted that "... the risk of lung cancer contributed to by asbestos exposure appears to be virtually independent of the age when that exposure took place and will be simply proportional to cumulative dose." (RCA Report, Vol.1, ch.5, p.294). The Panel is in general agreement with Doll and Peto's conclusion (1985) that the eventual risk of lung cancer is certainly not strongly related to age at first exposure to asbestos.

2.2.9 The RCA found that "latency periods are difficult to define or measure precisely, but on the basis of observed cases for asbestosis and asbestos-related cancers other than mesothelioma,

they are rarely less than 10 years and often more than 20 years." (RCA Report, Vol.1, ch.5, p.290) The RCA employed a delay time of 10 years in its modeling of the dose-response relationship between the relative risk of lung cancer and time since first exposure. It cited as support for this position illustrative data from the insulator workers' study (Selikoff, 1979) and from a study by Seidman (1979) (RCA Report, Vol.2, ch.10, p.451-3). The Panel's update found that all post-RCA epidemiological studies, with but two exceptions (Seidman, 1986; Acheson, 1984), reported no significant excess of lung cancers before 10 years from initial employment (Appendix A, pp.46). In one of the two exceptions (Seidman, 1986), 2 cases were found in the severe exposure category with less than 10 years of latency; in the other (Acheson, 1984), 17 cases (where 6.5 deaths were expected) were found in the 5-9 years from first exposure group. This evidence in total seems to support the statement that "little if any excess risk is produced for at least five years after first exposure, even under conditions of severe exposure, and the increase in risk caused by prolonged exposure at lower levels may not be detectable for 15 or 20 years" (Doll and Peto, 1985, pp.34-35). The Panel has decided that, for the purposes of eligibility rules, a latency period of 10 years should apply. It also emphasizes the pertinence of the Seidman and Acheson evidence with respect to benefit of doubt in case by case adjudication where latency is less than 10 years (see Section 3 of this Report).

2.2.10 In regard to measures of exposure, the staff review considered three indices (App. A, pp.46-48 and Table 3, pp.73-75):

- Intensity of exposure (viz. particles/ml, fibres/cc, or the qualitative categories of low, moderate and severe exposure);
- Duration of employment as a surrogate for duration of exposure;
- Total dose (viz. f/cc.years or mppcf.years measuring the product of exposure concentrations and duration of exposure).

The staff review found that many, but not all, pre- and post-RCA studies employing total accumulated dose for exposure reported excess risks in the lowest exposure categories examined.

2.2.11 The Panel found the following comments on the difficulties in assessing the quantitative effects of asbestos by Sir Richard Doll and Julian Peto (1985) to be both instructive and persuasive:

"First, asbestos is not a single chemical but a family of compound chemicals that have crystallised in nature as long thin separable fibres with some useful mechanical properties in common. Secondly, the biological effects are due partly to the chemical constitution of the material and partly to the physical configuration of the fibres, both of which vary with the type of asbestos that is used. Thirdly, the proportion of fibres with specific configurations vary with the ways in which the material is handled: they are different in mines and mills, in factories producing different end products, and in places where the end products are used in different ways. Fourthly, asbestos is commonly mixed with other materials. For example, it constitutes only some 5-20% of the ore that is mined, but it is progressively refined during milling to produce commercial grades. These also vary, the textile grades having the longest fibres and greatest purity. During product manufacture other materials may be added, as in making insulation board and asbestos cement. The presence of these materials may modify the effects of asbestos and increase the difficulty of assessing exposure."

"It follows that the biological effects of exposure to asbestos cannot be predicted by simple measures of the amount in the environment, but require a detailed specification of the mineralogical type and the number of fibres of different sizes and shapes. This last is hardly characterised at all by weighing the amount of dust in the air or by counting particles of all types, and is characterised only imperfectly by most of the methods for counting fibres that have been used in the past. And to add to these difficulties, we have insufficient knowledge of the mechanisms of human carcinogenesis to know precisely what quantitative measures are most appropriate for measuring the biological response."

"... Human evidence has little to contribute on the biological effects of fibres of different sizes, as human exposures have generally been to a wide range of sizes and environmental measures have seldom specified the mix with any precision."

[Effects on Health and Exposure to Asbestos. Doll,R. and Peto,J. London: Health and Safety Commission. HMSO, 1985]

2.2.12 On the basis of the staff review and of conclusions quoted above in Paragraph 2.2.11 on the difficulties in measuring asbestos exposure and assessing the quantitative health effects therefrom (Doll and Peto, 1985), the Panel has decided that numerical measures of intensity of exposure and of accumulated total dose should not be used as factors in the establishment of eligibility rules. Such numerical measures have little support in theory and the scarcity of records of past quantitative exposures defy their

application in practice.

2.2.13 With length of employment as the exposure measure, significant excesses of lung cancer risk appeared in conditions of severe exposure with less than 10 years of employment in one study (Peto, 1985), and with 2 or fewer years of employment in another (Newhouse, 1985). Where duration of employment was the sole measure of exposure, elevated risk appeared in 1-5 years of employment in several studies (Acheson, 1984; Seidman, 1986; Newhouse, 1985). In its consideration of this issue, the RCA found that "a dose-response relationship for lung cancer ... is best described by a linear non-threshold model or, on a graph, by a straight line through the origin" (RCA Report, Vol.1, ch.5, p.284). The Panel's review of the more recent epidemiological studies has led it to endorse this RCA position. In weighing all this evidence, the Panel determines that, under conditions of substantial exposure, excess risk can appear in as little as 2 years of employment.

2.2.14 Over what period of time have workplaces in Ontario existed in which workers were subjected to substantial asbestos exposures? Some insight into this matter can be provided by a review of the chronological development of asbestos regulations in Ontario. Historically, these regulations have included general legislative provisions for the health of workers in construction, mining and other fixed place industries, as well as specific legislative and non-legislative provisions applicable to asbestos exposure and use. Although dust control legislation has existed since 1884, no specific exposure limits for asbestos were established until 1947. At that time, the Ontario Ministry of Health began using the Threshold Limit Value (TLV) of 5 million particles per cubic foot (5 mppcf) as a criterion for the issuing of control orders for asbestos dust and in making recommendations to employers. This TLV was based on a recommendation by the American Conference of Governmental Industrial Hygienists (ACGIH) and remained the standard in Ontario until 1970. However, the TLV standard had no legal status and was only a guide to good practice. This TLV standard was usually presented in three forms: 1) as a time weighted average (TWA) over a normal 8 hour workday or 40 hour workweek; 2) as a short term exposure limit (STEL) averaging exposure levels over a 15 minute period during which this STEL value should not be exceeded more than 4 times during a workday;

and 3) as a ceiling value above which exposures are not permitted. The practice involved use of TWAs with the provision that STELs and ceilings never be exceeded.

2.2.15 The Factory, Shop and Office Building Act of 1913, which provided a legislative framework in the mining and industrial sectors for the control of asbestos once it was identified as a serious occupational hazard, was repealed in 1964 and replaced by The Industrial Safety Act. Under Ontario Regulation 196/64 of this Act, asbestos was for the first time specifically recognized as a health hazard. Industrial safety legislation was revised in 1971 and Regulation 259/72 provided more stringent regulation of asbestos in defined circumstances and also provided for medical examinations, labeling of containers and posting of notices.

2.2.16 Under the Construction Safety Act of 1962, protection by mechanical ventilation from hazardous exposure to noxious gas, fumes or dust was provided by regulation. Under this Act as well, Regulation 419/73 was passed in 1973 so severely limiting the use of asbestos in spray that it quickly disappeared as a practice in the construction industry.

2.2.17 Until 1970, the technique for measuring asbestos concentration involved the use of impinger sampling followed by the counting of particles using optical microscopy. Changes in international practice by the late 1960s resulted in the redefining of asbestos from simple particle counts to fibres longer than 5 microns and with a length to diameter ratio of at least 3.0. With this new definition and the continued use of counting fibres with an optical microscope, the Ontario Ministry of Health adopted the new standard of 5 fibres/cc recommended by the ACGIH in 1970.

2.2.18 By 1973, the Ontario Ministry of Health had abandoned the concept of TLVs as a reference criterion and had endorsed a guideline based on a time weighted average exposure limit of 2 f/cc for asbestos fibres greater than 5 microns in length as the target exposure level for Ontario industry. In 1975, the Occupational Health Branch of the Ontario Ministry of Health added a further provision limiting exposure to amphibole asbestos to 0.2 f/cc for fibres greater than 5 micrometres in length.

2.2.19 The Panel also gained some insight into the achievability during

the 1972-1976 period of the 2 f/cc Ontario standard from a study of asbestos levels in 5 Ontario asbestos handling plants and in about 100 other plants with similar practices (Rajhans, 1978). The authors stated that "no plants in this province who regularly use asbestos in their manufacturing operations have been able to keep their fiber levels below 2 for 100 percent of their measurements over a period of years. As a consequence, it is our opinion that the present methods of dust control as used in these plants are near their limit at 1-2 f/ml. We have no doubt that fiber levels of 0.5 f/ml and 0.1 f/ml can be achieved. The methods, however, will of necessity be different from those of local exhaust ventilation and the careful housekeeping practices presently used in the best Ontario plants. The order of magnitude increases in collection efficiency necessary to achieve 0.5 f/ml and 0.1 f/ml would only be obtained by totally enclosed processing, all wet processing or some similar technology."

2.2.20 Nevertheless, as late as 1975-1976, average concentrations as high as 12-15 f/cc were recorded in certain Ontario mines (OFL Submission to the RCA, January, 1981).

2.2.21 Under the Occupational Health and Safety Act of 1979, Ontario Regulation 570/82 was proclaimed in August, 1982 making asbestos a designated substance. O.Reg.570/82 applied to the mining, manufacturing and assembling industries, and to repair, alteration or maintenance processes involving machinery and equipment. This regulation provided a standard of 0.2 f/cc for crocidolite, of 0.5 f/cc for amosite, and of 1.0 f/cc for chrysotile.

2.2.22 On March 16, 1986, Ontario Regulation 654/85 was proclaimed and was applicable to non-fixed place industry (viz. construction, repair, alteration or maintenance of a building, or to every building which contained friable asbestos, etc.). It did not provide any quantitative standards but rather elaborated a number of stringent practices designed to "regulate by procedure".

2.2.23 With respect to determining conditions of substantial exposure therefore, the Panel notes that by 1973, the Occupational Health Branch of the Ontario Ministry of Health had adopted as its guideline the time-weighted average exposure limit of 2 f/cc for all types of asbestos as a criterion in issuing control orders

and in making recommendations to employers following air sampling. Further, in 1975, the Ministry of Health adopted a guideline of 0.2 f/cc for the amphiboles (RCA Report, Vol. 1, ch.3, pp.124-9). In this light, the Panel has decided that, for workers employed in Ontario industry before January 1, 1975, a factor of 2 years of employment should be used as the quantitative measure of exposure in an eligibility rule.

2.2.24 For post-1974 exposures, there is little guidance available from the epidemiological literature as to an appropriate minimum required duration of employment. However, the Panel would expect it to be longer than 2 years. The Panel notes that the proclamation of O.Reg.570/82 in August, 1982 made asbestos a 'designated substance' and provided a more stringent standard of 0.2 f/cc for crocidolite, of 0.5 f/cc for amosite, and of 1.0 f/cc for chrysotile. This standard remains in force today. Essentially therefore, the passage of the new regulation delineates a time frame from 1975 to about 1983 wherein risk to workers was, in an epidemiological sense, approaching non-detectability.

2.2.25 There is no reason to expect that with any carcinogen, lower exposure levels will result in longer latencies. Therefore, a 10 year latency would still be appropriate for claims qualifying for compensation. Given the likely induction period of lung cancer and the generally accepted 5 year period for lagging exposures [see the Panel's report on the Healthy Worker Effect, IDSP Report No. 3: July, 1988], the Panel has determined that a minimum of 5 years duration of exposure is appropriate for the 1975-1983 period of time in Ontario industries, but with the retention of a 10 year latency from time of first exposure. For a worker employed both before and after January 1, 1975 (and before January 1, 1983), the Panel simply recommends that a figure of 5 years of exposure be used, and that pre-1975 exposures be multiplied by 2.5 times before adding them to post-1974 exposure in order to determine eligibility under this criterion.

2.2.26 Of the factors which could be used to strike an appropriate eligibility rule for claimants for compensation for lung cancer from asbestos exposure in Ontario industries, the Panel is persuaded that the body of accumulated evidence can reasonably

support a quantitative measure of 10 years for the latency period of this disease; and a quantitative measure for exposure of at least two year's duration before 1975 (or five year's duration of employment between 1975 and 1983; or use of the aforementioned formula for employment before 1983 spanning the point in time of January 1, 1975). It was not persuaded that a rule which distinguishes fibre type or fibre size, or which specifies numerical values for age at first exposure could be supported in terms of its practicability of application by the Panel's review of the accumulated evidence. With respect to asbestos exposure from post-1982 employment, the Panel, bearing in mind the latency period of 10 years, considers that the matter of an eligibility rule should be considered in 1992-93.

2.2.27 The Panel has been persuaded of the superiority of schedules with restrictive language over their equivalent eligibility rules because the schedule approach triggers a rebuttable presumption and provides visibility in a clear and simple form to workers of the qualifying criteria for compensation for any given industrial disease. However, the Panel is unsure of the statutory underpinning for the use of restrictive language in schedules under the Ontario Workers' Compensation Act (see Paragraph 3.2.3 below). As a consequence, the Panel is presenting its recommendation for asbestos related lung cancer (and for the other cancers as well) in two forms: in a Schedule 3 form with restrictive language, and in an equivalent eligibility rule format. The Schedule 3 version of the lung cancer rule is as follows:

RECOMMENDATION 1: **THAT THE FOLLOWING ENTRY FOR ASBESTOS RELATED LUNG CANCER BE INSERTED INTO SCHEDULE 3 OF THE WORKERS' COMPENSATION ACT:**

DISEASE COLUMN	PROCESS COLUMN
PRIMARY CANCER OF THE TRACHEA, BRONCHUS OR LUNG DIAGNOSED AT LEAST 10 YEARS AFTER THE BEGINNING OF THE EMPLOYMENT IN THE PROCESS COLUMN.	PROOF OF EMPLOYMENT IN ONTARIO IN A PROCESS THAT INVOLVED: (A) A REPEATED OR CONSTANT RISK OF EXPOSURE TO ASBESTOS; OR (B) A REPEATED OR CONSTANT ACTUAL EXPOSURE TO

ASBESTOS;
AND THAT INVOLVED A DURATION OF AT LEAST:
(A) 2 YEARS BEFORE JANUARY 1, 1975; OR
(B) 5 YEARS BETWEEN JANUARY 1, 1975 AND JANUARY 1, 1983; OR
(C) 5 YEARS IN TOTAL DURATION DERIVED BY MULTIPLYING ALL PRE-1975 EMPLOYMENT BY 2.5 AND ADDING THIS TO EMPLOYMENT BETWEEN JANUARY 1, 1975 AND JANUARY 1, 1983.

2.2.28 In the above recommendation, the Panel distinguishes between actual exposure to asbestos and risk of exposure to asbestos in order to differentiate between employment in fixed as opposed to non-fixed place occupations. Employment where a fixed place asbestos control program was or should have been in place under Ontario regulations is deemed to have involved actual exposure. Because it has never been possible to measure actual exposure arising from non-fixed place employment, risk of exposure is the appropriate criterion.

2.2.29 Should it be found that there is insufficient statutory underpinning in the current Act for the Schedule 3 version shown in Recommendation 1 above, the Panel recommends the following equivalent eligibility rule:

ELIGIBILITY RULE 1: THAT CLAIMS ARISING FROM CANCERS OF THE TRACHEA, BRONCHUS OR LUNG AMONG ONTARIO WORKERS AND MEETING THE FOLLOWING CRITERIA BE COMPENSATED:

1. PROOF OF EMPLOYMENT IN ONTARIO IN A PROCESS THAT INVOLVED:
(A) A REPEATED OR CONSTANT RISK OF EXPOSURE TO ASBESTOS; OR
(B) A REPEATED OR CONSTANT ACTUAL EXPOSURE TO ASBESTOS;

AND THAT INVOLVED A DURATION OF AT LEAST:

(A) 2 YEARS BEFORE JANUARY 1, 1975; OR

(B) 5 YEARS BETWEEN JANUARY 1, 1975 AND JANUARY 1, 1983; OR

(C) 5 YEARS IN TOTAL DURATION DERIVED BY MULTIPLYING ALL PRE-1975 EMPLOYMENT BY 2.5 AND ADDING THIS TO EMPLOYMENT BETWEEN JANUARY 1, 1975 AND JANUARY 1, 1983.

2. **PRIMARY CANCER OF THE TRACHEA, BRONCHUS OR LUNG.**

3. **LATENCY PERIOD OF AT LEAST 10 YEARS (BETWEEN FIRST EMPLOYMENT IN ANY PROCESS DEFINED ABOVE AND THE DIAGNOSIS OF A PRIMARY LUNG CANCER).**

2.3 GASTROINTESTINAL CANCER

2.3.1 The RCA found a causal association between occupational asbestos exposure and cancers of the gastrointestinal tract (i.e. of the esophagus, stomach, colon and rectum). It pointed out that the association is less consistent and less marked (i.e. exhibiting lower standardised mortality ratios [SMRs] than, for example, lung cancer) when it has been found in cohort studies testing the association between asbestos and lung cancer, mesothelioma, and asbestosis (RCA Report, Vol.1, ch.5, pp.256-7). The staff review (App.A, pp.60-62) found that the current evidence continues to support the existence of a causal relationship. For reasons which are explored below, the Panel accepts the current evidence and makes the following finding:

FINDING 2: THE PANEL CONFIRMS THE EXISTENCE OF A PROBABLE CONNECTION BETWEEN CANCERS OF THE ESOPHAGUS, STOMACH, COLON AND RECTUM AND OCCUPATIONAL EXPOSURE TO ASBESTOS.

2.3.2 Factors which complicate the study of asbestos-related GI cancers include: varying case fatality rates (CFRs) for the different

cancer sites: the differences in observed effects from fibre types: the possibility of misclassification of death diagnosis: the levels of exposure in terms of both duration and intensity; and the treatment of latency. Epidemiological studies using mortality as the end point necessarily find fewer cases for diseases with low CFRs (defined as the proportion of cases which progress to death). Since GI cancers are a composite category, they include sites with lower CFRs (viz. rectal cancer) along with sites with higher CFRs (viz. esophageal cancer). This problem could be resolved through incidence studies but the literature is characterised by a paucity of such studies, the Danish study of asbestos cement industry workers (Raffn, 1987) being a notable recent exception. This latter study found a significant excess of "digestive organ" cancers (ICD-7 150-158) concentrated mainly in the stomach.

2.3.3 The RCA found that amphibole-exposed populations are more likely to exhibit clear dose-response relationships and elevated mortality rates for GI cancers than are chrysotile-exposed groups (RCA Report, Vol.1, ch.5, pp.256-7). The Panel, however, is of the view that this observation is more related to the fact that there appears to be a relationship between the excess relative risks for lung cancer and for the GI cancers. Thus, Doll and Peto (1985) have found, in their survey of a number of cohort studies, that the excess GI cancer risk is about 20% of that for lung cancer. A staff analysis of more recent asbestos related GI cancer data has provided confirmation of the Doll and Peto relationship (L'Abbé, March 12, 1990). If this finding is coupled with the observation in Paragraph 2.2.6 above that amphibole-exposed groups tend to show a greater health risk for lung cancer than do chrysotile-exposed groups, then it stands to reason that GI cancer risk in chrysotile studies will be lower. Also, given the problem identified in Paragraph 2.3.2 above that mortality studies fail to reveal all the GI cases and result in large sampling variability, the Panel is not surprised to find mortality studies of chrysotile-exposed workers showing little or no health risk for these cancers. Given this line of reasoning and others cited above on mixed fibre type exposures (Paragraph 2.2.7), the Panel does not consider fibre type to be a useful discriminator for the purposes of establishing standards for compensation for asbestos-related GI cancers.

2.3.4 Doll and Peto (1985) have raised the possibility of misclassifying other cancers, principally mesothelioma and lung cancer, as GI cancers. However, a recent comparison by Frumkin (1988) suggests that, while the argument remains plausible, no available evidence supports it with regard to gastrointestinal cancer.

2.3.5 The Panel has quoted extensively from a report by Doll and Peto (1985) in Paragraph 2.2.11 above on the difficulties in assessing the quantitative effects of asbestos. Small numbers of cases for GI cancers and the lack of comparability between studies prevent a clear evaluation of the relationship of exposure (whether considered as duration, or intensity or as accumulated dose) to risk. Also relevant is the fact that mortality studies cannot measure the incidence of non-fatal GI cancers. However, Frumkin (1988) has proposed an insightful scheme for addressing the measurement dilemma. He has observed that lung cancer risk is an effective surrogate measure of asbestos exposure circumstances. The RCA concluded that lung cancer risk is linearly related to exposure: and that there is no threshold of risk (i.e. the line graphing excess lung cancer relative risk against exposure passes through the origin). Frumkin uses this observed relationship to show that GI cancer risk increases with lung cancer risk.

2.3.6 Frumkin (1988) employs the doubling of lung cancer risk (i.e. an SMR of 200) as a discriminator for significant asbestos exposure. When used in conjunction with the assumption of a 20 year latency for GI cancers, this discriminator effectively identifies the excess risk for the GI sites. Frumkin's analysis is supported by the Panel's staff review (App.A, Table 7, p.88). The Panel therefore considers 20 years to be an appropriate latency period in its consideration of an eligibility rule for these cancers. In this context, the Panel notes that a number of different sites are involved with varying periods from diagnosis to death. Panel considers that this is pertinent with respect to benefit of doubt in case by case adjudication where latency is less than 20 years (see Section 3 of this Report).

2.3.7 For the reasons described in Section 2.2 above, the Panel has decided that two year's duration of employment is an appropriate criterion of exposure for an eligibility rule for lung cancer

(for workers employed before 1975). To find a comparable criterion for the GI cancers, the Panel has applied Doll and Peto's finding that excess GI cancer risk is about 20% of that for lung cancer. On the basis of this relationship, the Panel concludes that ten years' duration of employment is an appropriate factor for defining a quantitative measure of exposure for the GI cancers.

2.3.8 Given a 20 year latency for these cancers, exposures after 1974 should not result in GI malignancies before about 1994. A distinction for post-1974 exposures among workers would therefore be only of theoretical value. Moreover, it is an arguable proposition that GI cancer claims from asbestos exposure in the future will more likely arise among workers in non-fixed place industries. The RCA found that "there does appear to be some epidemiological evidence, indirect in nature, to support the notion that short, sharp bursts [i.e. high exposures of brief or intermittent duration] may carry a disproportionate health risk" (RCA Report, Vol.1, ch.5, p.303-6). The Panel finds that the accumulated evidence provides additional support for this RCA finding. The mechanism for carcinogenesis could well be that "excessive amounts of asbestos dust inhaled in any one period of time may overload the lung's clearance mechanism" as testimony before the RCA suggested (ibid, p.303). Nevertheless, health risk from such brief, intense exposures must be cumulative. The Panel notes that the epidemiological evidence is confined by necessity to studies of fixed place industries.

2.3.9 Absent records of asbestos concentrations, claimants who worked in conditions of intense occupational exposure will be denied the possibility of corroborating their employment histories. It is a known fact that in non-fixed place industries such as those involving building construction, repair, maintenance, alteration and demolition, fibre counts are not available. Whereas it is far easier to control exposures at source in fixed place industries, it is virtually impossible in non-fixed workplaces. In these latter industries, controls are by procedure; and the opportunities for direct exposure may be correspondingly greater especially when those procedures are relaxed. The Panel is also aware that the Regulation respecting Asbestos on Construction Projects and in Buildings and Repair Operations, made under the Ontario Occupational Health and Safety Act, did not come into

effect until March 16, 1986.

2.3.10 Given the foregoing considerations, including those which link GI cancer risk with lung cancer risk, the Panel recommends the placing of the following entry into Schedule 3 of the Act:

RECOMMENDATION 2: **THAT THE FOLLOWING ENTRY FOR ASBESTOS RELATED GASTROINTESTINAL CANCER BE INSERTED INTO SCHEDULE 3 OF THE WORKERS' COMPENSATION ACT:**

DISEASE COLUMN	PROCESS COLUMN
PRIMARY GASTROINTESTINAL (GI) CANCER, NAMELY CANCER OF THE ESOPHAGUS, STOMACH, COLON OR RECTUM, DIAGNOSED AT LEAST 20 YEARS AFTER THE BEGINNING OF THE EMPLOYMENT IN THE PROCESS COLUMN.	PROOF OF EMPLOYMENT IN ONTARIO IN A PROCESS THAT INVOLVED: (A) A REPEATED OR CONSTANT RISK OF EXPOSURE TO ASBESTOS; OR (B) A REPEATED OR CONSTANT ACTUAL EXPOSURE TO ASBESTOS; AND THAT INVOLVED A DURATION OF AT LEAST 10 YEARS.

2.3.11 As before, the Panel provides below an equivalent formulation of the above recommendation as an eligibility rule:

ELIGIBILITY RULE 2: **THAT CLAIMS ARISING FROM GASTROINTESTINAL (GI) CANCERS, NAMELY CANCERS OF THE ESOPHAGUS, STOMACH, COLON AND RECTUM, AMONG ONTARIO WORKERS AND MEETING THE FOLLOWING CRITERIA BE COMPENSATED:**

1. PROOF OF EMPLOYMENT IN ONTARIO IN A PROCESS THAT INVOLVED:
   (A) A REPEATED OR CONSTANT RISK OF EXPOSURE TO ASBESTOS; OR
   (B) A REPEATED OR CONSTANT ACTUAL EXPOSURE TO ASBESTOS;
   AND THAT INVOLVED A DURATION OF AT LEAST 10 YEARS.
2. PRIMARY CANCER OF THE ESOPHAGUS,

STOMACH, COLON OR RECTUM.

3. **LATENCY PERIOD OF AT LEAST 20 YEARS (BETWEEN FIRST EMPLOYMENT IN ANY PROCESS DEFINED ABOVE AND THE DIAGNOSIS OF A PRIMARY GI CANCER).**

2.4 LARYNGEAL CANCER

2.4.1 Cancer of the larynx is a relatively rare disease among men in developed countries, with about one case occurring for every 9 or so lung cancer cases (based on 1983 Ontario cancer incidence). Moreover, it has a low case fatality rate with about one death occurring from laryngeal cancer for every 30 deaths from lung cancer (1983 Ontario mortality statistics). Even more than in the case of GI cancers therefore, it is not surprising to find a paucity of mortality studies of a size large enough to identify excess risk from laryngeal cancer. Indeed, based upon 1983 Ontario mortality rates, a cohort mortality study would not be expected to find a single case of laryngeal cancer unless there were at least 30 lung cancer cases.

2.4.2 Occupational mortality studies may also underestimate risk if, for instance, CFRs for asbestos workers are affected by medical surveillance programs. Confounders of laryngeal cancer risk include tobacco and alcohol consumption since these lifestyle habits account for a large proportion of the overall risk for this disease. However, the Panel has not found any reason to suppose that asbestos workers drink more than average. Nor has a recent survey of the smoking habits of British asbestos workers revealed that this risk factor could account for more than 5% of any excess risk (Doll and Peto, 1985). Doll and Peto go on to suggest that misdiagnosis of other asbestos-related cancers, such as lung cancer and mesothelioma, for laryngeal cancer is unlikely and could not account for findings in case/control studies.

2.4.3 The Panel notes the existence of Ontario data (Finkelstein, 1988b) which indicate a significant excess of laryngeal cancer cases in a cohort of workers manufacturing asbestos containing friction products. This same cohort does not show a significant excess of lung cancer cases. There may be a qualitative difference between the nature of the exposures that induce

laryngeal as compared with lung cancer. It is possible that larger particles containing asbestos fibres were generated in this plant. And these particles were deposited in the upper airways and not inhaled in sufficient quantity into the bronchial tree so as to induce lung cancer.

2.4.4 The Panel is led to accept the evidence, therefore, as continuing to support a causal association between asbestos exposure and laryngeal cancer. As a consequence, it makes the following finding:

FINDING 3: THE PANEL CONFIRMS THE EXISTENCE OF A PROBABLE CONNECTION BETWEEN LARYNGEAL CANCER AND OCCUPATIONAL EXPOSURE TO ASBESTOS.

2.4.5 For reasons discussed above, the Panel does not accept the use of fibre type as a useful discriminator for the purpose of establishing an eligibility rule for laryngeal cancer. The Panel staff review for this cancer found that, for most cohort studies reporting latency, 20 or more years usually elapsed between first employment and the appearance of significant excess risk (App.A, pp.57-59). However, there were several notable exceptions (Selikoff, 1979; Rubino, 1979; Finkelstein, 1988b) including an Ontario case/control study of laryngeal cancer which reported that, of 23 subjects with occupational asbestos exposure, only 2 reported a period from first exposure of less than 10 years (Burch, 1981). The Panel has decided on balance that 15 years is the most appropriate value for establishing a minimum latency period for this disease. It is important to note that this latency period is derived mostly from mortality studies whereas in many cases the disease may be non-fatal. The Panel wishes to emphasize the pertinence of this observation with respect to benefit of doubt in case by case adjudication (see Section 3 of this Report).

2.4.6 There is no doubt that risk of laryngeal cancer from asbestos exposure is less than that from lung cancer. What is unclear is by how much it is less. Moreover, the paucity of large incidence studies prevents a clear determination of the dose-response relationship. Nevertheless, the staff review of mortality studies (App.A, Tables 6 and 7, pp.82-88) leads the Panel to consider that, absent contradictory evidence, risks for CI and

laryngeal cancers are comparable. Following the same line of reasoning as for the GI cancers therefore, it is reasonable and consistent to use a factor of ten years' duration of employment as a criterion of exposure in an eligibility rule for laryngeal cancer. The Panel notes that, with a minimum latency period of 15 years, post-1974 exposures among workers would only now be likely to begin to yield excess cancers. And arguably, laryngeal cancer claims are more likely in the future to arise among workers in non-fixed place industries.

2.4.7 On the basis of these considerations, the Panel recommends the placing of the following entry into Schedule 3 of the Act:

RECOMMENDATION 3: THAT THE FOLLOWING ENTRY FOR ASBESTOS RELATED LARYNGEAL CANCER BE INSERTED INTO SCHEDULE 3 OF THE WORKERS' COMPENSATION ACT:

DISEASE COLUMN	PROCESS COLUMN
PRIMARY LARYNGEAL CANCER DIAGNOSED AT LEAST 15 YEARS AFTER THE BEGINNING OF THE EMPLOYMENT IN THE PROCESS COLUMN.	PROOF OF EMPLOYMENT IN ONTARIO IN A PROCESS THAT INVOLVED: (A) A REPEATED OR CONSTANT RISK OF EXPOSURE TO ASBESTOS; OR (B) A REPEATED OR CONSTANT ACTUAL EXPOSURE TO ASBESTOS; AND THAT INVOLVED A DURATION OF AT LEAST 10 YEARS.

2.4.8 As before, the Panel provides below an equivalent formulation of the above recommendation as an eligibility rule:

ELIGIBILITY RULE 3: THAT CLAIMS ARISING FROM LARYNGEAL CANCER AMONG ONTARIO WORKERS AND MEETING THE FOLLOWING CRITERIA BE COMPENSATED:

1. PROOF OF EMPLOYMENT IN ONTARIO IN A PROCESS THAT INVOLVED:
   (A) A REPEATED OR CONSTANT RISK OF EXPOSURE TO ASBESTOS; OR
   (B) A REPEATED OR CONSTANT ACTUAL

EXPOSURE TO ASBESTOS;
AND THAT INVOLVED A DURATION OF AT LEAST 10 YEARS.

2. PRIMARY CANCER OF THE LARYNX.

3. LATENCY PERIOD OF AT LEAST 15 YEARS (BETWEEN FIRST EMPLOYMENT IN ANY PROCESS DEFINED ABOVE AND THE DIAGNOSIS OF A PRIMARY LARYNGEAL CANCER).

3. CASE BY CASE ADJUDICATION

3.1 THE BOARD'S INTERPRETATION OF BENEFIT OF DOUBT

3.1.1 From its very first Report of Findings, the Panel has provided advice to assist the Board in its exercise of the benefit of doubt provision embodied in Section 3(4) of the Act. Moreover, in both the gold mining and uranium mining reports (IDSP Report Nos. 1 and 6), the Panel has provided detailed supplementary material to assist adjudicators of claims for lung cancer among Ontario miners. The Panel has also recommended the approach first put forward by the RCA (RCA Final Report, Vol.3, ch.13, Sect.D, pp.765-7) concerning the interpretation of benefit of doubt. This recommendation (Recommendation No. 4, IDSP Report No. 1) is repeated below:

RECOMMENDATION 4: THAT IN EFFECTING CASE ASSESSMENTS, THE BOARD DRAW FROM ALL THE CIRCUMSTANCES OF THE CASE AND ALL THE EVIDENCE DISCOVERED BY OR PRESENTED TO IT EVERY REASONABLE INFERENCE IN FAVOUR OF THAT CLAIMANT; AND ACCEPT AS PROOF OF ANY FACT THAT THE CLAIMANT IS REQUIRED TO PROVE, ANY CREDIBLE EVIDENCE SUBMITTED BY HIM OR HER THAT IS NOT CONTRADICTED AND WHERE, IN WEIGHING ANY EVIDENCE SUBMITTED TO IT, ANY DOUBT EXISTS AS TO WHETHER THE CLAIMANT HAS ESTABLISHED HIS OR HER CASE, THE BOARD SHALL RESOLVE SUCH DOUBT IN FAVOUR OF THE CLAIMANT.

3.1.2 The Panel is disturbed by the persisting allegations that

eligibility rules serve as rules of exclusion and that claimants failing to meet the exact letter of any rule are denied compensation, notwithstanding the provisions of Section 3(4). The Panel is particularly concerned by allegations which have been drawn to its attention suggesting that no lung cancer claims among gold miners which did not meet fully the Board's policy for this industrial disease have been compensated.

3.1.3 The Panel considers that it is reasonable to surmise that, were the interpretation of Section 3(4) provided in Recommendation 4 consistently and properly made, at least some of these gold mining/lung cancer claims, albeit an unknown number, might have received compensation. For this reason, the Panel makes the following recommendation:

RECOMMENDATION 5: **THAT THE BOARD REVIEW ITS INTERPRETATION AND APPLICATION OF THE BENEFIT OF DOUBT PROVISION [SECT.3(4)] OF THE ACT RESPECTING CLAIMS FOR INDUSTRIAL DISEASE AND PLACE THE FINDINGS OF THIS REVIEW ON THE PUBLIC RECORD.**

3.2 ADJUDICATORY DISCRETION AND BARRIERS TO COMPENSATION

3.2.1 In the Panel's First Report on Asbestos, two recommendations called for the scheduling of asbestosis and mesothelioma in Schedule 4 of the Worker's Compensation Act. The Panel was careful in those recommendations to avoid the use of language which could set up artificial barriers to compensation, and which might allow the operation of unstructured discretion during adjudication.

3.2.2 For instance, the Panel called for an identical description to be placed in the "process" column of Schedule 4 for both these diseases. This description was: "any mining, manufacturing, assembling, construction, repair, alteration, maintenance or demolition process involving exposure to asbestos". Thus, the recommendations provided a list of industries in which the evidence indicated the likelihood of significant excess risk for these diseases from occupational asbestos exposures.

3.2.3 The Board has responded by distributing both diseases between

Schedules 3 and 4, and by inserting restrictive language in the Schedule columns. The Panel notes that the Board's legal capacity to use restrictive language in the Schedule columns is not explicitly authorized by the Ontario statute. In the British Columbia statute, by contrast, the use of restrictive language in Schedule columns has an explicit statutory base [British Columbia Workers' Compensation Act, RS Chap.437, Sect.6(4)(a)]. This legal question aside, the Panel is further concerned by the kinds of restrictions entered into the Ontario Schedules. Thus, for example, the Schedule 4 process entry for asbestosis requires "a reliable history of exposure to airborne asbestos fibres at a level approximating at least 25 fibres/cc-years". The same column entry refers among other processes to construction and demolition. It is misleading to include such processes in the same column that specifies a fibre count requirement when it is well known that fibre counts are not available for non-fixed place industry.

3.2.4 With respect to the language in both Schedules 3 and 4, the Panel must express its concern with the recurring phrase which requires a "reliable history" of asbestos exposure. This phrase is reminiscent of similar language in the Board's previous policy for all the asbestos-related cancers (i.e. lung cancer, mesothelioma, gastrointestinal cancers and laryngeal cancer). In those guidelines, the Board invariably referred to a "clear and adequate history" of occupational exposure to asbestos. In the past, the use of such language has apparently resulted in discretionary adjudicatory practices of questionable merit.

3.2.5 For example, the Panel has been given instances in which adjudicators have taken employment histories for claimants with over 30 years employment in asbestos-handling industries. In a number of these claims, the occupational histories were discounted to less than 10 years because the adjudicators required evidence of direct exposure to asbestos dust in preference to evidence of employment. In this way, claimants would fail to qualify for compensation according to a rule which required at least ten years of occupational asbestos exposure. However, the Board must be reminded that the epidemiological evidence for exposure upon which these policies were erected were based on duration of employment as the measure of asbestos exposure. Thus, the Board's restrictive language allows an

alleged discounting practice to be used by adjudicators to reject claims for compensation involving diseases which are counted on the basis of simple employment duration in epidemiological studies. Such allegations suggest the arbitrariness which can arise through the interpretation of policy couched in restrictive terms. They also contribute to the importance that the Panel attaches to Recommendation 5.

3.2.6 The Panel has considered the advisability of recommending the scheduling of lung, gastrointestinal and laryngeal cancer. It is cogniscent of the legal uncertainties concerning restrictive language in Schedule columns, and more especially the particular language which the Board chose to use in scheduling asbestosis and mesothelioma. Nevertheless, the Panel has decided to venture into these uncharted waters by providing recommendations to schedule with restrictive language for these cancers. It has also chosen to recommend eligibility rules corresponding to these recommendations to schedule in case the current wording of the Act does not allow this approach.

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April 18, 1990

MEMORANDUM TO: **THE WORKERS' COMPENSATION BOARD**

FROM: **LINDA JOLLEY, JOHN CHONG AND HOMER SEGUIN, MEMBERS OF THE INDUSTRIAL DISEASE STANDARDS PANEL**

RE: **DISSENT FROM THE MAJORITY SECOND REPORT ON CERTAIN ISSUES ARISING FROM THE REPORT OF THE ROYAL COMMISSION ON ASBESTOS**

We concur with the findings of the Panel confirming the existence of a probable connection between cancers of the trachea, bronchus, lung, esophagus, stomach, colon, rectum and larynx and occupational exposure to asbestos. We also concur with Recommendations 4 and 5. This dissent addresses Sections 2.2.25 and 2.2.26, 2.3.9 to 2.3.11, and 2.4.7 to 2.4.8.

The Panel is presuming that, because a guideline of 2 fibres per cubic centimetre was instituted in Ontario in 1972, compliance was achieved by 1975. The assumption, therefore, is that before 1975 fixed place exposures might average around 5 fibres per cubic centimetre, while after 1975, fixed place exposures would average below 2 fibres per cubic centimetre. This conclusion is based upon sampling data taken in five plants, one milling asbestos, two plants manufacturing brake linings and clutch facings, one plant manufacturing disc pads and one plant manufacturing asbestos-cement pipes and insulation. While the authors conclude that a fibre level of 2 was achievable and was being achieved on average in many of Ontario's asbestos industries, their data shows that both the milling and the asbestos-cement pipe producer were unable to keep all of their fibre levels below 2. Indeed, readings from the Johns-Manville Reeves Mine outside of Timmins in 1974 averaged 14.7 f/cc and reached levels of 75 f/cc and one reading of 225 f/cc. At the Matachewan Mine outside of Kirkland Lake, levels of 12 f/cc in March, 1976 and 43 f/cc in April, 1976 were recorded by one of the authors before that mine was closed by the government.

Even today, a Ministry of Labour Report on asbestos in the schools has indicated that many schools are not complying with that asbestos regulation, and the Ministry of Labour has admitted that their Designated Substances Regulations are difficult to enforce, so there is no reason to believe that there was widespread compliance with only a guideline that the Ministry of Labour itself indicated was unenforceable by law.

In Section 2.2.11, the Panel refers to a quote from Doll and Peto (1985) to describe the difficulties in assessing the quantitative effects of asbestos: "And to add to these difficulties, we have insufficient knowledge of the mechanisms of human carcinogenesis to know precisely what quantitative measures are most appropriate for measuring the biological response." And yet, behind the duration of employment requirements proposed is the assumption that 10 fibre years per cubic centimetre will produce an SMR of more than 200 or a relative risk of more than 2.0. While we agree that to formulate a rule requiring actual fibre counts is impossible due to the lack of adequate and proper monitoring, using employment duration as a surrogate ignores the difficulty described above.

In Section 2.2.13, the Panel confirms the finding of the RCA that the dose-response relationship between asbestos and lung cancer is "best described by a linear non-threshold model, or on a graph, by a straight line through the origin". This model establishes that every exposure above zero can contribute to these asbestos workers' risk of lung cancer. To limit the total burden of compensable lung cancer to only those workers who fall within a statistical excess based on insufficient knowledge of the connection between exposure and biological response, expressed with an exposure guideline based on the vagary of assumed compliance, is not only arbitrary but also fails to recognize the real contribution that asbestos makes to all of the lung cancer experienced by asbestos workers.

By basing the gastro-intestinal or laryngeal cancer risk on a multiple of the lung cancer risk, the same arguments about exposure criteria apply.

In Section 2.2.9, the Panel reports two studies that show significant excesses of lung cancer before ten years from the time of first exposure, and confirms Doll and Peto's statement that with severe exposures latency could be as little as five years.

As well, latency in these studies is the time from first exposure to asbestos to the date of death, not to the date of diagnosis at which time one of these asbestos victims could apply for compensation. Given average

survival rates of a year and a half for lung cancer and of much longer durations for gastro-intestinal cancer, it is inappropriate to use statistical latencies taken from mortality studies in this way.

Epidemiology is able to identify a probable connection between asbestos exposure and cancer of the trachea, bronchus, lung, gastro-intestinal tract and larynx. However, it cannot identify those individuals whose cancer is occupationally related and those whose cancer is not. Nor should compensation be limited only to those who fall within the statistical excess identified by latency and exposure criteria, based arbitrarily on assumed compliance with a guideline.

We do not believe that the Workers' Compensation Board has the legislative authority to implement Eligibility Rules into either Schedule 3 or 4, and therefore reject this option as presented by the majority on the Panel.

Nor do we believe that case by case assessment for those who just fall short of Eligibility Rules addresses the problem that workers have in bearing the burden of proof.

Since we believe that there is a probable connection between cancer of the trachea, bronchus, lung, esophagus, stomach, colon, rectum and larynx and occupational exposure to asbestos, it is appropriate for the Workers' Compensation Board to use Section 122(9) of the Workers' Compensation Act for the purposes of compensating those workers.

For these reasons, we as Panel Members recommend:

RECOMMENDATION: **THE BOARD SHOULD ENTER CANCER OF THE TRACHEA, BRONCHUS, LUNG, ESOPHAGUS, STOMACH, COLON, RECTUM AND LARYNX AND EXPOSURE TO ASBESTOS INTO SCHEDULE 3 OF THE WORKERS' COMPENSATION ACT, AND THAT CANCER OF THE TRACHEA, BRONCHUS, LUNG, ESOPHAGUS, STOMACH, COLON, RECTUM AND LARYNX SHALL BE DEEMED TO HAVE BEEN DUE TO OCCUPATIONAL EXPOSURE TO ASBESTOS, UNLESS THE CONTRARY IS PROVED.**

APPENDIX A

REPORT ON THE RELATIONSHIP BETWEEN OCCUPATIONAL ASBESTOS EXPOSURE AND MALIGNANT DISEASES

Dr. K.S. Yeung

Industrial Disease Standards Panel

July 5, 1989

REPORT ON THE RELATIONSHIP BETWEEN OCCUPATIONAL ASBESTOS EXPOSURE AND MALIGNANT DISEASES

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EXECUTIVE SUMMARY

1. This report provides updated epidemiologic information (to August, 1988) concerning the relationship between occupational exposure to asbestos and deaths from certain malignant diseases which will assist the development of appropriate eligibility rules for the adjudication of claims for compensation for these occupational diseases. The diseases included are lung cancer, mesothelioma, cancer of the larynx, and cancers of the esophagus, stomach, colon and rectum (i.e. the gastrointestinal tract).

2. The epidemiological evidence considered for each cancer site includes: the evidence for work-relatedness, the effects of industrial processes, of asbestos fibre types, of age at first exposure, of intensity and duration of exposure, and of disease latency.

3. The post-RCA (Royal Commission on Asbestos) epidemiologic literature has reinforced the causal relationship between occupational exposure to asbestos and excess lung cancer disease. The evidence linking more industries with this occupational disease now includes mining and milling, textiles, gas mask manufacturing, friction material and cement product industries, insulation and insulation products, general manufacturing of asbestos containing products, shipyard, electrochemical, sheet metal, plumbing and pipefitting industries.

4. Scientific evidence links all asbestos fibre types (including amosite, anthophyllite, chrysotile, crocidolite and tremolite) with excess lung cancer disease. The risk appears to be similar for men and women and not affected by the age of first exposure to asbestos. Most studies demonstrate increasing risk after 10 years following initial employment (the so-called latency effect). Higher intensities of exposure and longer durations of employment increase the risk of contracting this disease. When latency is adequate, increased risk has been demonstrated even at relatively low intensities of exposure. When intensity of exposure is high, short durations of employment of as little as 2 years have been shown repeatedly to cause excess deaths. Asbestos workers who smoke are much more at risk of lung cancer because of the synergistic effect between asbestos exposure and smoking.

5. The post-RCA epidemiologic literature has also reinforced the causal

relationship between occupational exposure to asbestos and mesothelioma. More asbestos-related industries have been found with deaths from mesothelioma. Evidence shows that all three fibre types of asbestos are causal agents of mesothelioma. However, chrysotile may be less potent than amosite or crocidolite, particularly with respect to causation of peritoneal mesothelioma.

6. The risk of mesothelioma is similar for both men and women and not affected by age at initial employment. Cases rarely appear before 10 years from initial employment and most cases appear 20 years from first employment (the latency effect). Longer durations of employment and higher intensities of exposure produce more deaths. A substantial number of mesotheliomas have been shown to occur among workers with low exposure and short durations of employment of less than 10 years. The risk is not affected by smoking habit. Some deaths from mesothelioma have been reported among family members of asbestos workers and contamination of the home by asbestos from the workplace is the likely cause.

7. Occupational exposure to asbestos has been linked to laryngeal cancer but the finding is not consistent. Large prospective morbidity studies incorporating adjustments for the strong confounding effects of tobacco and alcohol usage will help to resolve the issue unequivocally.

8. There is sufficient evidence to support the connection of occupational asbestos exposures with cancers of the gastrointestinal tract (including the esophagus, stomach, colon and rectum). Higher risks have been shown to occur with increasing time since first employment. Some studies have shown a greater number of excess deaths occurring with more intense exposures.

9. Four Ontario cohort studies have reported results consistent with other world scientific literature. They indicate excess risks for lung cancer, mesothelioma and gastrointestinal cancers among workers occupationally exposed to asbestos. The excess risk of lung cancer mortality was demonstrated in workers with just over 1 year of employment in 1 plant.

SECTION 1

INTRODUCTION

1.1 OUTLINE OF THE REPORT

This report is intended to provide updated epidemiologic information (to August, 1988) concerning the relationship between occupational exposure to asbestos and deaths from certain malignant diseases which will assist the development of appropriate eligibility rules for the adjudication of claims for compensation for these occupational diseases. The diseases included are lung cancer, mesothelioma, cancer of the larynx, and cancers of the esophagus, stomach, colon and rectum (i.e. the gastrointestinal tract). Issues of purely scientific interest (viz. exact dose-response models, carcinogenic mechanisms, etc.) are not addressed or addressed only briefly. For every finding of interest, the Royal Commission on Asbestos (RCA) conclusion is briefly stated and then updated information on the matter is provided. The updated material includes both new publications of epidemiological studies already considered by the RCA, and newer studies not appearing in the RCA evidentiary base. A separate section is devoted to the experience of Ontario asbestos workers as reported in four studies conducted by the Ontario Ministry of Labour.

As they did for the RCA, cohort studies have provided the major weight of evidence in this report. Cohort studies compare disease mortality or morbidity in a defined group of asbestos-exposed workers with that for a reference group, either the general population or another unexposed group of workers, by tracing the health experience of the defined cohort longitudinally forward in time from exposure to disease outcome. The measure of occupational risk usually employed in cohort studies is the Standardized Mortality Ratio [SMR] (or Standardized Morbidity Ratio: SMbR) defined as the ratio of the observed to expected numbers of deaths (or cases). The expected numbers are based on the mortality or morbidity experience of the matched reference group. All reported cohort studies (but one) were mortality studies of workers.

Another methodological approach to the determination of risk in occupational studies is the case-control method which traces the causal link backwards in time from disease outcome to occupational exposure. However, there are few available case-control studies involving occupational exposure to asbestos in part because asbestos exposure in the general population is a rare experience. Sometimes, case-control studies

are derived from the data in large cohort studies, and provide confirmation of the cohort findings freed from the influence of such artefacts as the healthy worker effect. In these so-called nested case-control studies, appropriate controls are drawn for the disease cases of interest by randomly selecting suitable controls from those cohort members matched by, for example, age, time since first hire, smoking habits, etc. Designing a case-control study with these matching criteria enables the investigator to control his analysis for the possible confounding effect of these factors.

SECTION 2

ON OCCUPATIONAL ASBESTOS EXPOSURE AND LUNG CANCER

2.1 CAUSAL RELATIONSHIP

The causal relationship between occupational exposure to asbestos and lung cancer is well recognized. According to established criteria for causality, most of the 24 cohort studies used by the RCA consistently demonstrated that occupational exposure to asbestos increases a worker's risk of dying from lung cancer. Since 1984, newer cohort studies of asbestos workers have reinforced the causal connection. In addition, several case-control studies for lung cancer, after adjusting for smoking habits, have identified occupational exposure to asbestos as an independent and significant risk factor (Damber, 1987; Schoenberg, 1987; Benhamou, 1988). Some questions have been raised concerning the degree of hazard posed by chrysotile fibres at very low levels of exposure and these will be addressed in the section on fibre types. Table 1 gives pertinent descriptions about these cohort studies together with their findings on lung cancer mortality.

2.2 EFFECT OF INDUSTRIAL PROCESS

The aforementioned cohort studies involved many industrial processes including: mining and milling; the manufacture of friction material, textiles, cement products, insulation products, gas masks and insulation; and shipyard work.

The excess risk of dying from lung cancer varied greatly among these industries. The RCA was concerned about differential hazards to workers, some industrial processes being exceptionally unsafe. Nevertheless, the RCA concluded that it was not possible to attribute the difference in risk to the difference in industrial processes alone even though, for instance, insulation manufacturing seemed much more dangerous than mining (RCA Report, Vol.1, ch.5: pp.224-231). This was because the asbestos fibre types varied by industry and process as did intensities of exposure.

As indicated in Table 1, a large number of both new and updated cohort studies have appeared in the literature since the RCA. Hodgson and Jones (1986) reported the largest ever retrospective cohort study, including 31,150 workers in England and Wales employed in various asbestos-related

industries. No estimate of fibre exposure was made but duration of employment was used as a surrogate for cumulative dose. For those employed in the same industry for more than 10 years, insulation workers were most at risk of lung cancer (SMR=347), while board and paper industry workers were least at risk (SMR=77). Unfortunately, no information on fibre type was available. Several single industry cohort studies have appeared showing variation in the risk of lung cancer mortality within the same industry. These differences resulted largely from differences in cohort definitions, types of asbestos fibre and intensities of exposure.

Important additions to the post-RCA literature have come from investigations of several industries that are not exclusively asbestos-related. Some demonstrated elevated lung cancer risks for their workers. Kolonel (1985) reported a significantly increased lung cancer risk for shipyard workers employed no less than 15 years and with latency of no less than 30 years (SMR=170), although no excess risk was demonstrated for the cohort at large. Hilt (1985) reported a large risk (SMR=616) in a small cohort (N=97) of electrochemical workers heavily exposed to asbestos; while Cantor (1986) reported a significant excess risk (Proportional Mortality Ratio or PMR=141) in a cohort of over 7,000 plumbers and pipe-fitters. Michaels (1988) investigated a cohort of sheet metal workers with substantial exposure to asbestos and found an increased lung cancer SMR of 186.

2.3 EFFECT OF FIBRE TYPE

In Canada, the asbestos mined or used in manufacturing falls into two main fibre groups, serpentine and amphiboles, which differ both structurally and chemically. Only chrysotile appears as a serpentine fibre. It is usually white, curly, pliable and easily spun. The major industrial uses of chrysotile are in textiles, cement products, flooring and friction materials. There are several types of amphiboles, the most common being amosite and crocidolite. Other amphiboles such as tremolite and anthophilite are infrequently mined and used but often appear as contaminants in ores. Amosite is grey, yellow or dark brown; crocidolite is blue. Amphiboles are straight, needle-like and fairly spinnable. They are used in the production of pressure pipes and insulation products. Asbestos use worldwide comprises approximately 90% chrysotile, and 9% amosite and crocidolite combined (RCA Report, Vol.1, ch.2: pp.75-93).

Chrysotile exposures have been considered less hazardous to workers with

respect to lung cancer risk. However, the RCA (RCA Report, Vol.1, ch.5, pp.236-244) concluded that there was conflicting evidence on the issue since asbestos exposure in a predominantly chrysotile setting led to large excesses of lung cancer deaths in some plants even though, in general, chrysotile mines and plants had lower lung cancer SMRs. The mixed influence of industrial process, intensity of exposure and fibre type render the interpretation of the relative contribution from each individual factor difficult. The RCA noted that animal studies have demonstrated that all three fibre types (crocidolite, chrysotile and amosite) have similar carcinogenic potentials when directly implanted onto the target organ. One way to reconcile the relative contribution to health risks from fibre type and industrial process is by interpreting the differences in terms of fibre dimensions. It is postulated that straight, thin fibres of between 5-8 um have greater pathogenic potential because they can be readily airborne and inhaled to travel down to body sites and penetrate organ linings; they are less efficiently destroyed by the body's immune system. In general, straight thin fibres are more abundant in amphiboles, but industrial processes such as in the textile industries can split fibres to produce more and thinner components regardless of the type of fibre used. The RCA adopted this explanation and concluded that the pathogenicity of asbestos is primarily a function of fibre dimensions and that amphiboles are more likely to have higher pathogenicity because of their size and structure (RCA Report, Vol.1, ch.5, p.269).

The effect of tremolites was discussed by the RCA but the available information at the time was not sufficient to allow conclusions on the relative potency of tremolites in lung cancer causation. However, a completed investigation of a vermiculite mine and mill in the U.S. heavily contaminated with fibrous tremolites has reported a statistically significant risk (SMR=223), similar to the risks reported for chrysotile and crocidolite mining (Amandus, 1987).

Two studies (Ohlson, 1985; Gardner, 1986) involving cohorts of asbestos cement manufacturing workers exposed to low levels of chrysotile (below 2 f/ml, on the average, for both studies) demonstrated no increase in risk. In both studies, the turnover rates for workers were high (40% worked for less than two years in the Ohlson cohort; and 52% worked for less than 1 year in the Gardner cohort) which fact could explain the relatively low reported risks. For both studies, separate analyses were performed on groups with different latencies, durations of employment and exposure levels, but no group exhibited significant excess lung cancer mortality.

The most important report on this issue involved two cement manufacturing plants in New Orleans (Hughes, 1987). The general study results were previously published but a new report reassessing exposure levels has yielded additional information on fibre types. The two cement plants, with similar exposure levels, industrial processes, duration of employment for workers and with similar proportions of smokers, exhibited different lung cancer risk estimates 20 years or more after initial employment. The plant handling mostly chrysotile had a lower risk than the one handling both chrysotile and crocidolite, (SMRs of 114 and 144 respectively). However, at similar durations of employment and levels of cumulative exposure, workers for the latter plant exposed only to chrysotile did not experience a lower risk than did their co-workers in the same plant who handled both chrysotile and crocidolite. Comparisons of lung cancer risk for various levels of duration of employment, cumulative exposure and fibre type are presented in Table 2. The authors concluded by stating that "although the across study comparisons of Doll and Peto suggested greater risks of lung cancer from exposure to amphiboles compared with chrysotile alone, firm conclusions on this issue cannot yet be drawn".

2.4 EFFECTS OF AGE AT FIRST EXPOSURE AND GENDER

The RCA concluded that for adult workers, the age at first exposure to asbestos has no effect on excess lung cancer risk (RCA Report, Vol.1, ch.5: pp.290-295).

Most recent studies have not investigated the effect of age at first exposure separately from duration of employment and latency. Of those which have, Hughes (1987) and Peto (1985) found no effect. Newhouse (1985) found no effect in men (SMRs of 280 and 240 for men first exposed before age 25 or from age 25+ respectively) but a pronounced effect for women (SMRs of 1,160 and 480 for women first exposed before age 25 or from age 25+ respectively). Alies-Patin (1985) showed that the lung cancer risk was particularly high for male French asbestos cement manufacturing workers who started work before age 25 when their duration of employment was no less than 20 years, but the number of deaths in that group was only 5.

The number of reported female workers and their duration of employment in all cohorts were smaller than for men. When analyzed separately, the two gender groups generally showed a similar response to exposure as indicated

by elevated lung cancer risk.

2.5 LATENCY OF THE DISEASE

Using models of carcinogensis as well as epidemiologic evidence, the RCA (RCA Report, Vol.1, ch.5, pp.290-295) decided that latency for lung cancer, i.e. time from initial exposure to clinical manifestation of disease, is over 10 years and often more than 20 years.

Most post-RCA publications reported findings with longer periods of time since first hire (typically 10-20 years) in order to allow for the latency effect. However, two studies including an update of the Paterson, New Jersey, study (Seidman, 1986) and another by Acheson (1984) have both demonstrated that, when the intensity of exposure was sufficiently high, lung cancer risk increased significantly within 5-10 years following initial exposure. Both studies involved exposure to amosite. The Seidman study had only 2 deaths with less than 10 years of latency from lung cancer (0.42 expected) among those with more than 100 f/cc.y exposure. But the Acheson study was based on 17 deaths (6.5 expected) in the latency group of 5-9 years. Other studies that investigated latency did not find a significant excess before 10 years from initial employment. An updated report on lung cancer mortality in the Acheson cohort (Gardner, 1988) showed that, with an addition of 6 years of followup, the same cohort yielded 36 additional lung cancer deaths (SMR=215) over and above the 57 reported deaths during the first 34 years (SMR=176).

2.6 DOSE-RESPONSE RELATIONSHIP

'Dose', i.e. the extent of exposure, has been estimated in three ways:

1) by measuring dust or fibre concentrations in the air of work sites (in units of mppcf for millions of particles per cubic foot, or f/cc for fibres per cubic centimetre), or simply as low, medium or high exposures;
2) by duration of employment in conditions of dust exposure; and
3) by multiplying dust level and duration of employment together to form a composite dust index or measure of total dose (in units of mppcf.y for millions of particle years per cubic foot, or f/cc.y for fibre years per cubic centimetre).

Neither 'duration of employment' nor 'dust level of work site' alone is

an adequate measure of dose, and the composite index regards the effect of time and dust level as interchangeable (e.g. 10 f/cc for 5 years is equivalent to 5 f/cc for 10 years). The RCA considered three major issues in dose-response relationships for asbestos exposure and lung cancer: the threshold effect (Is there a safe level of exposure below which no detectable excess of lung cancer deaths occurs?); the pattern of the relationship (i.e. a linear, sigmoid or other form); and the effect of peak exposures as characterized by high intensity and short duration. It was concluded that the most likely relationship between occupational exposure to asbestos and lung cancer is linear and without threshold, i.e. risk increases with the lowest dose of exposure and continues to increase with increasing exposure at a constant rate (RCA Report, Vol.1, ch.5, pp.283-284). The RCA considered that intermittent, high exposures may carry a disproportionately higher risk. However, available information was not adequate to settle the issue definitively. As a consequence, the RCA recommended that workers be protected from such exposures so as to minimize possible risk (RCA Report, Vol.1, ch.5, pp.305-306).

New and updated studies exploring risk by exposure level have emerged since the RCA. These studies provide information on dust concentrations which enable the further examination of issues of threshold and dose-response.

Studies with small numbers or showing no excess lung cancer deaths yield little information on questions of dose/response. For these reasons, discussion is provided only on studies with 30 or more observed lung cancer cases or deaths and in which a significant increase in lung cancer risk was demonstrated in at least one exposure level subgroup. Table 3 summarizes the findings. When total dose was used as the exposure measure, most studies demonstrated excess risks in the lowest exposure level examined. Risk increased with increases in exposure in all but 1 study (McDonald, 1984). However, with similar exposure levels, different studies often yielded different SMRs. For example, at levels of about 20-40 mppcf.y, SMRs of 156, 304, 105 and 200 were reported (McDonald, 1983a, 1983b, 1984; Hughes, 1987).

Duration of employment is a crude surrogate for exposure level since the actual numbers of inhaled fibres or particles could vary between jobs and over time. Nonetheless, in the absence of other measures, it is widely used and it can clarify the relative importance of duration and intensity of exposure. Most reports have shown increased lung cancer risk when duration of employment was less than 10 years. Significant increases were

found generally in studies reporting a high overall risk (Acheson, 1984; Seidman, 1986; Newhouse, 1985). Only 2 studies provided estimates stratified by intensity and duration of exposure. Peto (1985) showed that at exposure intensities of 400 p/ml or more, a significant increase of risk was found among workers with less than 10 years of employment. Newhouse (1985) showed that with "severe" exposure, risk increased significantly in workers with 2 or less years of employment. When duration of employment was the sole measure of dose, the risk estimates varied between studies. Therefore, duration of employment cannot adequately measure the level of exposure or the level of risk. To illustrate, SMR risk estimates for 1-5 years of employment ranged from 220 to 829 (in studies by Acheson, 1984; Seidman, 1986; and Newhouse, 1985) whereas much lower risks of 161 and 159 were reported only with more than 20 years of employment (in McDonald, 1980, 1983a).

An extensive treatment of dose-response relationships in the post-RCA literature can be found in a report for the Environmental Protection Agency (Nicholson, 1986). He concluded that "the accumulated data suggest that the excess risk of death from lung cancer from asbestos exposure is proportional to the cumulative exposure (i.e. the duration times the intensity) and the underlying risk in the absence of exposure".

2.7 INTERACTION WITH SMOKING

Information concerning the interaction effect of asbestos exposure and smoking on lung cancer risk has come from three major sources: Berry (1972), Hammond (1979), and McDonald (1980). All three studies indicate that smoking and asbestos exposure increase a worker's risk of dying from lung cancer multiplicatively (RCA Report, Vol.1, ch.5, pp.295-300). With an estimated relative risk (RR) of lung cancer among smokers vs non-smokers of about 11, and a RR among asbestos exposed vs non-exposed of about 5, the combined risk of smoking and asbestos exposure would be about 55 times that for non-smokers without exposure to asbestos [see Enterline (1983) for an analysis of data drawn from the Selikoff (1979) study]. The RCA has noted that the lung cancer risk from asbestos exposure is the same for smokers and for non-smokers. Since smokers have a much greater background risk of dying from lung cancer, the absolute number of lung cancer deaths attributable to asbestos would necessarily be much larger for smokers than for non-smokers.

Most new reports have not included information on workers' smoking status.

Some have attempted to compare the proportion of smokers in their studies to the proportion in a reference population in order to estimate the extent of bias on the estimation of lung cancer risk. In general, if the proportion of smokers is higher in the observed cohort than in the reference population, the lung cancer risk would tend to be overestimated, and vice-versa. For those studies that included smoking information, a prevailing problem in testing any model of interaction between smoking and asbestos exposure was the fact that almost no non-smoking lung cancer cases could be found. Nevertheless, Hilt (1985) showed that the combined effect of smoking and asbestos was multiplicative. He estimated the lung cancer RR from smoking to be 5.8 among the non-asbestos exposed; the RR from asbestos exposure to be 4.3 among smokers; and the combined RR for smoking, asbestos-exposed workers compared to non-smoking, non-exposed persons to be 25.2. On the other hand, Berry (1985) suggested that the effects of asbestos exposure are greater among non-smokers than among smokers (thus suggesting a submultiplicative model for the combined risk). The combined information from these studies leads to the conclusion that asbestos exposure and smoking have a synergistic effect on lung cancer risk (i.e. the joint effect of these factors is greater than the sum of their individual effects).

2.8 CONCLUSIONS

Recent epidemiologic evidence reinforces the finding that occupational exposure to asbestos is a cause of lung cancer. The issues of industrial process and fibre type have not been completely clarified, so that further investigation is still needed to assess the joint and independent effects of these two factors on lung cancer mortality. Nevertheless, the list of industries in which opportunities for asbestos exposure has led to excess lung cancer risk has grown since 1984. Studies continue to appear linking all fibre types (amosite, anthophyllite, chrysotile, crocidolite and tremolite) with excess lung cancer risk. In general, age at first exposure appears to be weakly related to lung cancer risk, although two recent studies do report some age-related effects. Small numbers of women in most reported studies prevent a definitive statement on gender-specific effects, but the evidence generally supports a similar response for both men and women to occupational exposure to asbestos. In general, excess mortality could be demonstrated 10 years from the time of initial exposure, but there is also evidence that a significant excess could occur 5-9 years following first exposure. There is solid evidence that, under certain conditions such as intense exposure, excess mortality follows

brief exposures of less than 2 years. New evidence continues to support a hyperadditive model of lung cancer risk, so that the relative risk (RR) of dying from lung cancer for smoking asbestos workers is more than the sum of the corresponding RR for smoking and that for asbestos exposure. This means that smokers are, in absolute numbers, more at risk of lung cancer from asbestos exposure than are non-smokers.

SECTION 3

ON OCCUPATIONAL ASBESTOS EXPOSURE AND MESOTHELIOMA

3.1 CAUSAL RELATIONSHIP

The causal relationship between occupational asbestos exposure and mesothelioma is clear and definite (RCA Report, Vol.1, Ch.2, p.100). Some agents other than asbestos, such as zeolites and wood pulps, have been shown to cause mesothelioma in humans but the number of cases produced is relatively small (Pelnar, 1986).

Mesothelioma is a rare disease, its incidence in the 1978-82 period being no higher than 5 cases per 100,000 population per annum in any registry reporting area (Table 4). The significance of occupational relatedness appeared in the 1950s when the incidence of pleural mesothelioma rose among North American males but not among females. Since then, male incidence has steadily increased suggesting a strong occupational component in the disease's etiology (McDonald, 1986).

Mesothelioma deaths have been reported in most post-RCA studies (Table 5). One case-control study on mesothelioma (Schenker, 1986) demonstrated that, after adjusting for age and date of death, railroad workers regularly exposed to asbestos were 21.4 times more likely to contract mesothelioma than their unexposed fellow workers.

3.2 ANATOMICAL SITES

Mesothelioma primarily occurs in the mesothelial tissue lining the pleural and peritoneal cavities. Most investigators have reported more pleural than peritoneal mesotheliomas (Table 5), excepting Selikoff who found 112 cases of peritoneal and 63 cases of pleural mesothelioma in American insulators. More peritoneal mesotheliomas have been found when the type of fibre was either amosite or crocidolite. After an examination of the evidence, the RCA concluded that it is likely that there is a differential effect on anatomical sites by different fibres (RCA Report, Vol.1, ch.5, p.252).

3.3 EFFECT OF INDUSTRIAL PROCESS

As in the lung cancer studies, the incidence of mesothelioma has varied for different industrial processes. The RCA highlighted the important effect of industrial process but pointed out that other factors such as fibre types and intensity of exposure are also important in determining the risk of mesothelioma (RCA Report, Vol.1, ch.5, pp.224-231).

Post-RCA material has contributed additional evidence to support the RCA conclusions and has added to the list of industries involved. Mesothelioma deaths have now been reported among railroad workers (Ohlson, 1984; Mancuso, 1988), sheet metal workers (Michaels, 1988), plumbers and pipefitters (Cantor, 1986) and shipyard workers (Kolonel, 1985).

3.4 EFFECT OF FIBRE TYPE

In the light of existing evidence, the RCA concluded that asbestos from all types of fibre are capable of inducing mesothelioma. However, the reported number of mesotheliomas caused by chrysotile alone is low. Chrysotile has rarely been reported to cause peritoneal mesothelioma and may be less hazardous than amphiboles in causing mesothelioma of the pleura (RCA Report, Vol.1, ch.5, pp.252-256).

Since 1984, new and updated reports have generally supported the RCA conclusions (Table 5). Moreover, a recent study has demonstrated the occurrence of 14 cases of pleural mesothelioma among 181 railroad workers exposed to chrysotile alone (Mancuso, 1988). One study of a crocidolite mining cohort in Australia (Armstrong, 1988) showed a low peritoneal to pleural mesothelioma ratio (1:32) thus casting doubt on the hypothesis that a higher incidence of peritoneal mesothelioma is associated with exposure to amphiboles. However, this study had over 20% lost to followup and its conclusion requires a cautious acceptance. A nested case-control study in New Orleans (Hughes, 1987) showed that, after matching for age and time since first employment and after adjusting for duration of employment, working in areas with crocidolite exposure elevated the risk of contracting mesothelioma among asbestos workers already exposed to chrysotile. In a commentary, Dunnigan (1988) suggested that chrysotile alone did not cause mesothelioma; and the fact that mesothelioma occurred among chrysotile miners could be explained by contamination of the mines by amphiboles such as tremolites. He cited as support lung tissue studies principally by Churg (1984, 1988) which showed higher levels of amphiboles

but not chrysotile in the lungs of mesothelioma cases when compared to lung tissue samples from controls.

This commentary has provoked a number of responses from prominent investigators in the field based on both epidemiological and clinical lung burden studies. McDonald (1988) supported the general thrust of Dunnigan's conclusion and emphasized the importance of the tremolite factor. However, he also pointed out that lung burden studies have major limitations and uncertainties:

> 1) Fibers present in lung tissue at death, at varying periods after first or last exposure, do not necessarily reflect the past, qualitatively or quantitatively;
> 2) Lung tissue studies depend on the availability of post mortem and surgical material, both of which are liable to many types of selection bias. This problem is compounded by the need for similar tissue from comparable referents;
> 3) Inhaled and retained fibres are not evenly distributed throughout the lungs...;
> 4) The significance of fibres in lung tissue at death will depend on the carcinogenic mechanism....

He concluded that "lung burden studies provide just one more element in the jigsaw of causation but do not necessarily outweigh other types of evidence." Churg (1988) refuted the conclusion drawn by Dunnigan on his work. He pointed out that "chrysotile itself does not accumulate in human lung to any great extent, particularly compared to amphiboles. It is very important to understand this point, because otherwise one can easily conclude that only amphibole is causing disease [...] What these data do indicate is that the chrysotile content of the lung may be a very poor marker of the amount of chrysotile exposure." On the issue of tremolite contamination, he said: "The published data are, therefore, consistent with the idea that tremolite is the agent of chrysotile-induced mesothelioma in man, but they are no more than consistent. The number of cases (especially cases in which pulmonary mineral content has been analyzed) upon which this information is based is very small and the possibility still remains that chrysotile fibre itself in sufficiently large doses is a mesothelial carcinogen". Other scientists (Kogan 1984, Sluis-Cremer 1988, Lilienfeld 1988, Roggli 1988, Davis 1988, Becklake 1988, Craighead 1988) have expressed an opinion on this issue. Although no consensus is apparent, there seems to be a general agreement that chrysotile is a less potent mesothelial carcinogen than amphiboles but exposure to chrysotile, particularly when heavy, could also cause mesothelioma and whether the real agent is or is not the contaminant

tremolite remains a contentious issue. Doll and Peto (1985) reviewed animal studies, lung burden studies and epidemiologic studies earlier, and their conclusion was that "it is not practicable to remove tremolite from chrysotile for commercial purpose and any distinction between the effects of chrysotile and tremolite may, therefore, be considered academic...". This conclusion appears still valid today.

3.5 EFFECTS OF AGE AT FIRST EXPOSURE AND GENDER

Age at first exposure to asbestos is important to the extent that it correlates with latency period as the RCA has pointed out (RCA Report, Vol.1, ch.5, pp.290-295).

There have been relatively few women in asbestos industries, the largest number having been employed in the manufacture of friction material and gas masks during the the Second World War. The number of mesotheliomas among women in gas mask manufacturing was high in almost all reported cohorts.

No other significant additional information pertaining to the effects of age and gender have emerged since the RCA.

3.6 LATENCY OF THE DISEASE

The RCA concluded that, in general it takes a relatively long time (over 20 years) for mesothelioma to manifest itself clinically. Moreover, the incidence increases with latency (RCA Report, Vol.1, ch.5, pp.290-295).

Most of the post-RCA reports have supported this conclusion, the shortest reported latency being 13 years after initial exposure (Kolonel, 1985) (see Table 4).

3.7 DOSE-RESPONSE RELATIONSHIP

The number of reported cases of mesothelioma is so small, even in the largest cohorts, that it is not possible to examine dose-response relationships between asbestos exposure and mesothelioma to the same extent as one can with lung cancer. Based on available information, the RCA concluded that the risk of dying from mesothelioma increased with

'dose', as measured by duration of exposure and intensity of exposure. The RCA also concluded that there was no safe threshold in the causal relationship. Mesothelioma could occur, even with brief exposures, provided that the latency period is adequate (RCA Report, Vol.1, ch.5, pp.286-290).

Most post-RCA reports have demonstrated that relatively low exposures [less than 30 f/cc.y (Finkelstein, 1984); as little as 48 mppcf.y (Enterline, 1987)] produce mesothelioma. Other studies (Hughes, 1987; Newhouse, 1985) have shown the appearance of mesothelioma following a sufficient latency period in workers with short durations of employment. This is consistent with the assumption of no safe threshold. A nested case-control study (Peto, 1985) showed that workers with at least 10 years of employment in high exposure areas had an extremely high risk (odds ratio of 14.7) when compared to workers with less than 10 years of employment.

An extensive treatment of the subject of dose-response relationships for mesothelioma can be found in Nicholson (1986).

3.8 INTERACTION WITH SMOKING

The RCA concluded that asbestos exposure and smoking do not have an interactive effect on the risk of mesothelioma, i.e. both smokers and nonsmokers have the same risk of developing the disease from asbestos exposure (RCA Report, Vol.1, ch.5, pp.301-302).

Berry (1985) compared mesothelioma death rates among smokers, ex-smokers and non-smokers in an asbestos factory. In men, the death rate was higher among ex-smokers than smokers (477 versus 201 deaths per 100,000 population), while no cases were found among non-smokers. In women, non-smokers had the highest rate (539 per 100,000) while ex-smokers and smokers had similar rates (266 and 245 per 100,000 respectively). They agreed with McDonald (1980) that "it seems improbable that tobacco smoking is an etiological factor of any importance in this disease".

3.9 FAMILY CONTACTS

Some clusters of mesothelioma among families of asbestos workers have been reported. Case series studies have also shown that a number of family

members of asbestos worker have contracted mesothelioma.

Based on this evidence, the RCA concluded that cohabitation with an asbestos worker is the likely cause of mesothelioma in non-occupationally exposed persons, and recommended that household members of asbestos workers who contract mesothelioma be compensated (RCA Report, Vol.3, ch.12, pp.715-716).

After the RCA, Lynch (1985) published a report on studies of families with mesothelioma which concluded that the evidence is consistent with the presence of host susceptibility among these families. This does not refute the postulate that exposure to asbestos in the households of asbestos workers is the cause of the manifestation of the disease since many cases of household contacts involved wives of asbestos workers who are not genetically related.

3.10 CONCLUSIONS

Recent epidemiologic information continues to reinforce the finding that occupational exposure to asbestos is the most important cause of mesothelioma. There is some evidence that, while chrysotile, amosite and crocidolite are capable of causing pleural mesothelioma, amosite and crocidolite seem to be more efficient in causing peritoneal mesothelioma. No evidence indicates any variation in incidence when age at initial exposure and gender are considered. However, as development of the disease usually requires a long latency period (over 20 years), initial exposure at younger ages would provide a higher chance for the disease to develop. Mesothelioma has been shown to appear often when exposure was low (less than 48 f/cc.y) and when duration of employment was brief (less than 2 years). There is clear evidence that smoking does not play a role in causing the disease. Cases of mesothelioma have been reported among family members of asbestos workers.

SECTION 4

ON OCCUPATIONAL ASBESTOS EXPOSURE AND CANCER OF THE LARYNX AND GASTROINTESTINAL TRACT

4.1 ASBESTOS AND CANCER OF THE LARYNX

The RCA report briefly mentioned that some studies have implicated asbestos as a cause of cancer of the larynx (RCA Report, Vol.1, ch.2, p.102).

Cancer of the larynx is a relatively rare disease. The 1983 incidence rate among men in Ontario was 7 per 100,000 while the corresponding incidence for lung cancer was 65.4 per 100,000. Case fatality from laryngeal cancer (i.e. the proportion of cases which progress to death from the same disease) is low as well. Laryngeal cancer mortality in 1983 among men in Ontario was 1.8 per 100,000 population compared with 54.4 per 100,000 for lung cancer mortality (Clarke, 1985).

Many factors, some possibly related to employment status, may influence the prognosis of the disease (e.g. early detection, accessibility of treatment and general health). For example, if early detection of laryngeal cancer among asbestos workers through medical surveillance improves their chances of survival, then this fact alone may offset a marginal increase in the mortality risk of this disease from asbestos exposure. As a consequence, mortality studies of laryngeal cancer (or, for that matter, of any low case-fatality disease) may not identify the real causal relationship between asbestos exposure and development of this disease. The true risk may be more accurately estimated by incidence studies, especially where the case-fatality rate is low and there is reason to believe that there may be differences (viz. early detection) between exposed and non-exposed groups. In addition, tobacco and alcohol use have been identified as strong risk factors for laryngeal cancer. Without understanding the extent to which disease outcome is affected by these two factors, the true effect of asbestos on risk of the disease could not be estimated fully.

Case-control studies of laryngeal cancer have investigated incidence cases rather than deaths and in this regard are more appropriate designs to study causal relationships between asbestos exposure and this disease. However, using case control methods has its disadvantages: a) substantial exposure to asbestos is rare in the general population which fact reduces

the number of exposed cases and controls; and b) self-reporting bias of persons diagnosed with cancer may incline the respondent to over-report or recall adverse exposures including asbestos. Three post-RCA case-control studies (Elwood, 1984; Olsen, 1984; Brown, 1988) have reported results incorporating adjustments for confounding factors such as tobacco and alcohol use. Elwood compared 154 laryngeal cancers patients with an equal number of matched controls drawn from the same hospital and reported that there was no association between occupational exposure to asbesto and cancer of the larynx. Olsen compared 326 cases with population controls and found a marginally significant odds ratio of 1.8. Similarly, Brown reported an odds ratio (OR) of 1.46 (95% confidence interval: 0.98-2.18) after comparing 183 laryngeal cancer cases with 250 matched population controls. A significant trend in risk (p=0.024) was demonstrated when the data were stratified by exposure levels: low intensity of exposure yielded an OR of 1.2; medium an OR of 1.5; and high intensity of exposure an OR of 2.8. However, no trend was found when subjects were grouped by number of years exposed (less than 5 years: OR=1.3; 5-14 years: OR=2.2; and 15 or more years: OR=1.4).

Table 6 shows the data from asbestos worker cohorts reporting laryngeal cancer results. Many of these studies reported no laryngeal cancer deaths. Others reported deaths only from major causes and remarked that no excess deaths from laryngeal cancer were observed without giving the actual number of deaths from this disease. Because of the low death rate from laryngeal cancer, cohort studies that reported less than 30 cases of lung cancer are not likely to have reported any deaths from laryngeal cancer unless the lung cancer (or all cause) SMR is extremely high. Therefore, negative findings from these studies do not imply that there is no effect. Studies that reported over 30 cases of lung cancer should yield at least 1 death from laryngeal cancer if asbestos exposure has similar effects on lung cancer and on laryngeal cancer deaths. Of the 28 studies that reported fewer than 30 cases of lung cancer (Tables 1 and 6), 15 reported statistically significant lung cancer SMRs, while only 2 studies reported a significant excess of laryngeal cancer. Robinson (1979) observed 6 deaths (SMR=316) from laryngeal cancer but no excess of lung cancer deaths was observed (SMR=105). Finkelstein (1988b) reported 4 deaths (SMR=667) from laryngeal cancer but he stated that none of the 4 men worked in an asbestos-using department. This suggests that the excess of laryngeal cancer deaths in these two cohorts may have been due to reasons other than asbestos exposure. Of the 26 cohort studies that reported over 30 cases of lung cancer, 8 studies showed higher than expected numbers of deaths from laryngeal cancer (not all of which were

significant). Of these 8, 1 reported 11 cases with a significant SMR of 234 (Selikoff, 1979); another (Seidman, 1986) reported 7 cases with a marginally significant SMR of 192 ($0.05 < p < 0.10$). Eighteen studies reporting at least 30 lung cancer cases did not report excess laryngeal cancer deaths. Of these 18, 4 studies (Berry, 1983; Newhouse, 1985; Hodgson, 1986; and Hughes, 1987) had over 100 cases of lung cancer. A cohort study of incidence (Raffn, 1987) has reported 14 cases of laryngeal cancer with 8.44 cases expected ($p=0.07$).

The fact that most cohort studies have not reported significant excess deaths from laryngeal cancer does not provide evidence against a causal relationship between laryngeal cancer and occupational asbestos exposure. This is because the number of deaths or disease cases was small and because no adjustments were made for major confounding factors such as tobacco and alcohol use. Tobacco and alcohol usage have been demonstrated to be strong risk factors for laryngeal cancer. Burch (1981) estimated the odds ratios for heavy users of tobacco and alcohol to be 23.7 and 8.0 respectively. Since, therefore, the excess mortality is small and dependent on the smoking and drinking habits of each cohort, the observed effect of asbestos exposure on laryngeal cancer could change in either direction. For example, in case-control studies which adjusted for tobacco and alcohol use, the odds ratio decreased from 1.75 to 1.0 in one study (Hinds, 1979) while it increased from 1.6 to 2.3 in another (Burch, 1981), and no change was observed in the remaining two (Olsen, 1984; Brown, 1988). Most observed SMRs for laryngeal cancer deaths in the cohort studies are well below 200. It is not possible to predict the direction of the effect that would result from adjustment for tobacco and alcohol use in these studies.

In most cohort studies that reported latency for laryngeal cancer, very few cases occurred before 20 years from first employment (2 cases in the Selikoff cohort (1979); 2 cases in the Rubino cohort (1979); and 1 case in the Finkelstein cohort (1988b). Dose-response relationships were investigated but not demonstrated by McDonald (1980), and later by Liddell (1984) in the Quebec cohort.

It is biologically plausible that asbestos fibre inhalation could cause neoplastic changes in the larynx just as it causes neoplastic changes in the lung although not all lung carcinogens have been found to be laryngeal cancer carcinogens. Together with results from some case-control studies, this suggests that occupational exposure to asbestos may contribute modestly to the production of laryngeal cancer.

4.2 ASBESTOS AND CANCER OF THE GASTROINTESTINAL TRACT

The RCA has concluded that, in spite of the scarcity of evidence, the causal association between asbestos exposure and cancer of the esophagus, stomach, colon and rectum is highly likely. However, amosite and crocidolite are likely to be more carcinogenic to the gastrointestinal (GI) tract than chrysotile (RCA Report, Vol.1, ch.5, pp.256-257). There has not been sufficient information to evaluate the dose-response relationship and possible interactions with other risk factors.

Based upon reported incidence and mortality rates for GI cancers (Clarke, 1985), the approximate case fatality rates vary from 39% for rectal cancer to 100% for cancer of the esophagus. As for laryngeal cancer, therefore, mortality studies may not adequately estimate the causal relationship between asbestos exposure and GI cancers.

For cohort mortality studies, a number of new reports have been published since 1984 with somewhat inconsistent findings. The largest cohort study involving 31,550 men (Hodgson, 1986) failed to detect any excess deaths of cancer in all sites of the GI tract. The larger subcohort of the study conducted by Peto et al. (1985) also did not show an elevated risk. Nevertheless, a number of other studies have shown excess deaths from cancers of the GI tract (Newhouse, 1985; Seidman, 1986; Enterline, 1987; Armstrong, 1988). One cohort study of incidence also showed a significant increase in GI cancer, mainly concentrated in stomach cancer (Raffn, 1987).

Most recent studies reported mixed fibre types and therefore yielded little information on the issue of the relative hazards of different fibre types. One study comparing mortality in two plants, one of which used only chrysotile while the other handled both chrysotile and crocidolite (Hughes, 1987), failed to demonstrate a higher mortality from GI cancer in both plants. Two studies on cement workers who handled only chrysotile showed conflicting results, one involving a slight decrease in mortality (Gardner, 1986), the other a slight increase (Ohlson, 1985). Nevertheless, with the aforementioned exceptions, a general pattern has emerged indicating that, for larger cohorts and particularly those which demonstrate excess lung cancer mortality, excess GI cancers are also apparent.

Dose-response relationships have been examined in only a few studies with large numbers of cases (Newhouse, 1985; Peto, 1985; Seidman, 1986; Hughes, 1987). Increasing risk with increasing intensity of exposure (as measured by fibre years per cc [f/cc.y]) was demonstrated by Seidman (1986); while Newhouse (1985) only used low versus severe exposure contrasts. The other two studies (Peto, Hughes) failed to demonstrate a dose-response relationship. An updated matched case-control analysis of the Quebec cohort by Liddell (1984) showed significant increased risks for cancers of the esophagus and stomach in exposures over 3,000 f/ml.y (Odds Ratio = 4); and significantly increased risk for cancers of the colon and rectum in exposures over 1,000 f/ml.y (Odds Ratio = 2).

Within any given study, different sites in the GI tract did not always have increased or decreased risks. For instance, in plumbers and pipefitters (Cantor, 1986), the number of deaths from cancer of the stomach was elevated, but cancers of the colon and rectum were below expected numbers. This finding was duplicated for workers in the study of a cement product plant involving both chrysotile and crocidolite (Hughes, 1987). Since numbers of deaths for each site are small and many of the studies did not give information for each separate site in the GI tract, it remains difficult to distinguish the difference in effect of occupational exposure of asbestos on different sites.

Concerning latency for cancers of the gastrointestinal tract among asbestos workers, most cohort studies have reported some deaths mainly for extended times since first employment of at least 10 years, and usually 20 years (Table 7). Reported numbers of deaths and corresponding risk estimates for shorter latency periods were nearly always lower because workers whose occupational experience fell into such categories were usually younger and had shorter durations of employment. In general, reported risks increased with latency period.

Miller (1978) reviewed the case for GI cancers and concluded that there was in fact a causal relationship. Doll and Peto (1985) suspect that misclassification of lung cancer and mesothelioma deaths as deaths from GI cancers is the cause of the apparent excess of these cancers, and that no real causal relationship exists. Two reviews (Morgan, 1985; Levine, 1985) suggest that a conclusive answer will require more studies. Others have tried to resolve the inconsistencies in reported results. Edelman (1988) contends that excess mortality has only been demonstrated in North American cohorts the results of which, he suggests, have been subject to confounding factors such as dietary patterns. However, he provides no

substantiation for this contention. Frumkin (1988) showed that studies which demonstrated excess lung cancer mortality (SMR > 200) have typically shown elevated risk for gastrointestinal cancers also.

4.3 CONCLUSIONS

Current scientific evidence suggests that there may be a modest effect from asbestos exposure on laryngeal cancer risk. However, due to low incidence and case-fatality rates from this disease, the prevalence of strong confounding factors, as well as a scarcity of exposure in the general population, conclusive evidence could only come from large prospective morbidity studies that properly adjust for the effects of confounders.

The current evidence supports the existence of a causal relationship between occupational asbestos exposure and cancers of the gastrointestinal tract. Although mortality studies do not fully reflect the risk of contracting these cancers, dose-response relationships reported in some studies strengthen the evidence for a causal relationship with asbestos exposure.

SECTION 5

THE EXPERIENCE OF ONTARIO ASBESTOS WORKERS

There have been a number of asbestos manufacturing plants in operation in Ontario with workers under surveillance. By 1980, the RCA reported that the total number of asbestos workers under surveillance by the Ministry of Labour's Occupational Health branch was over 1,900 (RCA Report Vol.2, ch.6, p.320-321). Dr. Murray Finkelstein of the Ontario Ministry of Labour has conducted epidemiological studies on 4 of these Ontario asbestos plants. These studies provide crucial information for assessing the health hazards of asbestos to Ontario workers.

Table 8 briefly describes the 4 plants and the design of the Finkelstein studies. The asbestos plants manufacture insulation and friction products. All three major fibre types (chrysotile, crocidolite and amosite) have been used in these plants. Exposure levels were generally higher before 1960. However, accurate exposure estimates were not usually available.

All 4 studies had similar designs. Plant workers were identified by company records which provided information on age, sex, year of hire, type of work and duration of employment on each job for each worker. The study cohort would include all workers who had more than background exposure to asbestos. The cohort was then traced by matching names and dates of birth of workers with deaths recorded in data bases such as the Canadian Mortality Data Base maintained by Statistics Canada. Numbers of deaths in the cohort from all causes and from specific causes were then compared with the provincewide mortality to obtain estimates of risk. In 3 of the 4 plants, nested case-control studies were conducted by using controls selected randomly from the same work force.

Except for the Bendix study (Finkelstein, 1988b), all studies demonstrated elevated lung cancer risk caused by asbestos exposure (Table 9). Workers in Holmes (Finkelstein, 1987b) appeared to have the highest risk (SMR=606) although there were only 4 deaths involved. These findings are consistent with other large studies (Acheson, 1984; Seidman, 1986) reporting higher risk among workers manufacturing insulation products with amosite. The large number of reported lung cancer deaths in the Johns-Manville cohort (Finkelstein, 1988a) enabled investigation of the effects on risk of duration of employment and latency. It was shown that excess deaths from lung cancer appeared among workers exposed for over 1 year. A significant

dose-response trend was demonstrated showing elevated risk at 1-10 years of employment (Odds Ratio = 1.8) and the excess risk was most prominent among workers exposed for over 10 years (OR=4.5). Most deaths occurred 20 years after initial exposure.

Johns-Manville workers also suffered the highest number of deaths from mesothelioma (Table 10). Among those exposed to asbestos, there were 31 recorded cases, about 70% of which were pleural mesotheliomas. Excess death from gastrointestinal cancer was demonstrated among workers with over 1 year of employment and with 20 years from initial employment (SMR=194). Only the Bendix study reported deaths from laryngeal cancer (4 cases). Finkelstein concludes:

> There was a cluster of 4 deaths from cancer of the larynx among Bendix workers. This cluster is difficult to interpret. None of these men worked in an asbestos-using department, although 2 of them may have had direct contact with asbestos dust in their respective capacities as millwright and member of the clean-up crew. In addition, lung cancer rates are usually elevated more in asbestos-exposed workforces than are laryngeal cancer rates, the opposite of the situation that was found here. (Finkelstein, 1988b)

The lack of significant lung cancer risk in the Bendix study could have been a result of intrinsic study limitations. Company records could have excluded some exposed workers. Detailed information on work site and dust levels was not available to correctly assign exposure levels to individual workers. Therefore, it is possible that the lung cancer risk of Bendix workers has been underestimated. Moreover, the moderate increase in lung cancer deaths (SMR=124) is not statistically significant at the 5% level because the small size of the study population prevented the detection of a moderate increase in risk. Recall that, for the Quebec mining cohort (McDonald 1980), the risk of lung cancer death for the entire exposed cohort was about the same as for the Bendix workers (SMR=125) and was statistically significant because of the larger sample size. Small numbers also affected the conclusions which could be drawn from the Bendix case-control study. With either 12 cases or 18 (depending on the latency category), a misclassification of work site or of duration of employment for a few cases would substantially affect the risk estimates (see Kleinbaum, 1982). Nonetheless, the findings of this study are consistent with findings in two other larger studies of the friction material industry. One showed no effect (Berry, 1983) while the other (McDonald, 1984) showed a significantly increased overall risk for which, however, the excess deaths were mainly concentrated among workers employed for less

than 1 year. Neither study detected any dose-response relationship. In considering the relative hazards of different industrial processes, the RCA noted the low risks reported in cohorts of friction product workers (RCA Report Vol.1, ch.5, pp.182-188) but underlined the fact that, for both cohorts (Berry, 1983; McDonald, 1984), the exposure levels were extraordinarily low. No reliable information on historical exposure levels of Bendix workers is available. The only measurements were taken after 1975 and were reported to be low at levels below 2 f/ml.

In conclusion, the Ontario data demonstrates that Ontario asbestos workers have had similar health effects to those of other asbestos workers. The smaller work forces in Ontario prevented the detection of moderate increases in risk. However, when assessed in conjunction with results from larger studies, the Ontario evidence demonstrates that occupational asbestos exposure causes lung cancer, mesothelioma and gastrointestinal cancer among exposed workers. However, information on laryngeal cancer risk is extremely limited since post-RCA Ontario reports involved only mortality studies. Risk is higher for those with over 10 years of employment but is also demonstrated among workers with shorter employment duration (1-10 years). Most deaths occurred 20 years after the workers entered the plants suggesting that additional asbestos-related deaths will probably occur in the coming years.

TABLE 1

LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	FIBRE TYPES[1]	LOCATION	NUMBER[2]	FOLLOWUP PERIOD	LUNG CANCER OBS.	LUNG CANCER SMR	ALL CAUSES OBS.	ALL CAUSES SMR	LATENCY YRS
MINING AND MILLING:									
McDonald (1980)	ch	Canada	10,939#[3]	1926-75	230	125*[3]	3,291	109	20+
Rubino (1979)	ch	Italy	952	1946-75	11	106+[3]	332	155*	-
Nicholson (1979)	ch	Canada	544	1961-77	28	252*	178	111	20+
@[3]Armstrong (1988)	cr	Australia	6,505#	1943-80	91	264*	820	153*	-
@Amandus[4] (1987)	tr	USA	575	1970-81	20	223*	161	110	-
@McDonald[5] (1986)	tr	USA	406	1963-83	23	245*	165	117	-

NOTES:

1 Re fibre types:
- am amosite;
- an anthophyllite;
- ch chrysotile;
- cr crocidolite;
- tr tremolite.

2 Number of workers followed are all male unless otherwise indicated.

3 Symbols:
- # denotes that 10% or more of the cohort was lost to follow up.
- * denotes a two-tailed statistical estimate significant at the 5% level.
- + denotes subcohort with significant increase in lung cancer mortality.
- @ denotes cohorts not included in RCA or which have updated reports since the RCA.

4 Cohort of vermiculite miners exposed to tremolite as a contaminant.

5 Both the Amandus and McDonald studies were of workers in the same mine.

TABLE 1 (CONT'D.)

LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	FIBRE TYPES	LOCATION	NUMBER	FOLLOWUP PERIOD	LUNG CANCER OBS.	LUNG CANCER SMR	ALL CAUSES OBS.	ALL CAUSES SMR	LATENCY YRS
TEXTILE:									
McDonald (1983a) (Pennsylvania)	ch, cr	USA	4,137	1938-77	53	105+	895	109	20+
McDonald (1983b) (S.Carolina)	ch	USA	2,543	1938-77	59	200*	570	127*	20+
Dement[6] (1983)	ch	USA	1,261	1940-75	35	315*	308	150*	-
@Peto (1985)	ch, cr	UK I: male (>=20 yrs service)	145	1933-83	20	361*	123	181*	-
		II: female (>=10 yrs service)	283	"	4	211	49	99	-
		III: male	3,211	"	132	131*	1,113	114*	-
Robinson (1979)	ch, am,cr	I: male	2,722	1940-75	49	136*	912	123*	-
		II: female	554	"	14	824*	128	145*	-
GAS MASK MANUFACTURING:									
@Acheson (1982) (Blackburn, Eng.)	ch	UK	female 570	1951-80	7	145	177	138*	10+
@Acheson (1982) (Leyland, Eng.)	cr	UK	female 757	1951-80	15	241*	219	118*	10+
Jones (1980)	cr	UK	female 951#	1941-78	12	190*	166	?	-

NOTES: Re fibre types: amosite (am); anthophyllite (an); chrysotile (ch); crocidolite (cr); tremolite (tr). Number of workers followed are all male unless otherwise indicated. # denotes that 10% or more of the cohort was lost to follow up. * denotes a two-tailed statistical estimate significant at the 5% level. + denotes subcohort with significant increase in lung cancer mortality. @ denotes cohorts not included in RCA or which have updated reports since the RCA.

6 The study cohort was the same as in McDonald (1983b).

TABLE 1 (CONT'D.)

LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	FIBRE TYPES	LOCATION	NUMBER	FOLLOWUP PERIOD	LUNG CANCER OBS.	LUNG CANCER SMR	ALL CAUSES OBS.	ALL CAUSES SMR	LATENCY YRS
@Wignall (1982)	cr	UK female	523	1951-77	10	273*	133	96	10+
McDonald (1978)	cr	Canada (m+f)	199	1939-75	8	200*	43	-	-
FRICTION MATERIAL:									
Berry (1983)	ch, cr	UK I: male	7,474	1942-80	143	103	1,339	98	10+
		II:female	3,708	"	6	53	299	91	10+
@Finkelstein (1988b) (Windsor)	ch	Canada	1,657	1950-85	15	124	186	105	-
McDonald (1984) (Connecticut)	ch,cr, an	USA	3,641	1930-77	73	149*	803	109	20+
CEMENT PRODUCTS:									
@Hughes (1987)	ch	USA I:	1,898	1942-82 (Plant I)	47	114	477	91	20+
	ch,cr	II:	3,594	1936-82 (Plant II)	107	144*	874	95	20+
@Gardner (1986)	ch	UK I: male	1,514	1941-84	35	92	384	94	-
		I: female	659	"	6	142	102	92	-
@Ohlson (1985)	ch	Sweden	1,176	1951-82	11	122	220	103	20+
Thomas (1982)	ch,cr	UK	1,592	1936-77	28	85	261	107	15+
@Alies-Patin (1985)	ch,cr	France	1,506	1940-82	12	217*	206	92	-

NOTES: Re fibre types: amosite (am); anthophyllite (an); chrysotile (ch); crocidolite (cr); tremolite (tr). Number of workers followed are all male unless otherwise indicated. # denotes that 10% or more of the cohort was lost to follow up. * denotes a two-tailed statistical estimate significant at the 5% level. + denotes subcohort with significant increase in lung cancer mortality. @ denotes cohorts not included in RCA or which have updated reports since the RCA.

TABLE 1 (CONT'D.)

LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	FIBRE TYPES	LOCATION	NUMBER	FOLLOWUP PERIOD	LUNG CANCER OBS.	LUNG CANCER SMR	ALL CAUSES OBS.	ALL CAUSES SMR	LATENCY YRS
@Finkelstein (1988a)	ch,cr	Canada	3,166	1950-85	49	168*	408	104*	-
@Albin (1984)	mixed	Sweden	598	1951-80	12	183*	172	109	20+
@Raffn (1987) (Incidence Study)	mixed	Denmark	7,970	1928-84	162	180*	-	-	15+
INSULATION PRODUCTS:									
@Finkelstein (1987a)	ch	Canada	324	1929-82	6	162	57	93	-
@Finkelstein (1987b)	am	Canada	107	1956-87	4	606*	13	142	-
@Acheson (1984)	am	UK	4,820	1947-80	57	196*	333	111	-
@Gardner (1988)	same cohort with additional followup			1981-86	36	215*			
@Seidman (1986)	am	USA	820	1941-82	111	541*	593	167*	5+
GENERAL MANUFACTURING:									
@Weiss (1977)	ch	USA	264	1945-74	4	93	66	61*	-
@Enterline (1987)	ch,cr, am	USA	1,074	1941-80	77	271*	944	124*	retired
@Newhouse (1985)	mixed	UK I: male	2,886	1933-80	158	250*	818	134*	10+
		II: female	694	"	37	740*	274	176*	10+
@Nicholson (1976)	mixed	USA (m+f)	689	1969-71	27	321*	199	148*	20+

NOTES: Re fibre types: amosite (am); anthophyllite (an); chrysotile (ch); crocidolite (cr); tremolite (tr). Number of workers followed are all male unless otherwise indicated. # denotes that 10% or more of the cohort was lost to follow up. * denotes a two-tailed statistical estimate significant at the 5% level. + denotes subcohort with significant increase in lung cancer mortality. @ denotes cohorts not included in RCA or which have updated reports since the RCA.

TABLE 1 (CONT'D.)

LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	FIBRE TYPES	LOCATION	NUMBER	FOLLOWUP PERIOD	LUNG CANCER OBS.	LUNG CANCER SMR	ALL CAUSES OBS.	ALL CAUSES SMR	LATENCY YRS
INSULATION:									
Selikoff (1979)	ch,am	Canada & USA	17,800	1967-76	486	460*	2,271	137*	-
		USA (subset)	632	1943-76	93	699*	478	145*	-
@Newhouse (1985)	ch,cr,am	UK	1,369		38	355*	157	152*	10+
@Elmes (1977)	mixed	Belfast	162	1940-75	27	540*	122	165*	-
ANY ASBESTOS WORK:									
@Hodgson (1986)	unspec.	UK	31,150	1971-81					
		I: employed before 1969			157	130*	897	89*	-
		II: employed after 1968			29	108	231	84*	-
@Woitowitz (1986)	unspec.	Wt.Germany							
		I: exposed from 1972-77 (m+f)	3,070	1977-82	26	170*	185	95	-
		II: exposed before 1972 (m+f)	655	"	12	462*	71	175*	-
OCCUPATIONS WITH PROBABLE ASBESTOS EXPOSURES:									
SHIPYARD:									
@Kolonel (1985)	mixed	USA	7,971	1950-79					
		exposed:	5,191	"	61	117+	668	71*	-
Rossiter (1980)	mixed	UK	6,292	1947-78	84	70	1,043	104	-

NOTES: Re fibre types: amosite (am); anthophyllite (an); chrysotile (ch); crocidolite (cr); tremolite (tr). Number of workers followed are all male unless otherwise indicated. # denotes that 10% or more of the cohort was lost to follow up. * denotes a two-tailed statistical estimate significant at the 5% level. + denotes subcohort with significant increase in lung cancer mortality. @ denotes cohorts not included in RCA or which have updated reports since the RCA.

TABLE 1 (CONT'D.)

LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	FIBRE TYPES	LOCATION	NUMBER	FOLLOWUP PERIOD	LUNG CANCER OBS.	LUNG CANCER SMR	ALL CAUSES OBS.	ALL CAUSES SMR	LATENCY YRS
ELECTRO-CHEMICAL:									
@Hilt (1985)		Norway		1953-80					
	I: lightly exposed		190		5	200	75	107	-
	II: heavily exposed		97		8	616*	63	134*	-
SHEET METAL:									
@Michaels (1988)	unspec.	USA	407	1976-86	48	186[p7]*	?		-
PLUMBING AND PIPEFITTING:									
@Cantor (1986)	unspec.	USA	7,121	1960-79	636	141[p]*	7,121	-	-
RAILROAD WORK:									
@Ohlson (1984)	unspec.	Sweden	3,442	1951-80	37	116+	925	82*	20+

NOTES: Re fibre types: amosite (am); anthophyllite (an); chrysotile (ch); crocidolite (cr); tremolite (tr). Number of workers followed are all male unless otherwise indicated. # denotes that 10% or more of the cohort was lost to follow up. * denotes a two-tailed statistical estimate significant at the 5% level. + denotes subcohort with significant increase in lung cancer mortality. @ denotes cohorts not included in RCA or which have updated reports since the RCA. p denotes that Proportional Mortality Ratios (PMRs) were used (see Monson, 1987: pp. 86-87).

[7] p denotes that Proportional Mortality Ratios (PMRs) were used (see Monson, 1987: pp.86-87).

TABLE 2

LUNG CANCER MORTALITY IN THE NEW ORLEANS ASBESTOS CEMENT MANUFACTURING PLANTS 20 YEARS OR MORE AFTER INITIAL EXPOSURE

Duration of Employment	Plant I Chrysotile Only	Plant II Chrysotile & Crocidolite[1]	Plant II Chrysotile Only[2]	Plant II Chrysotile & Crocidolite[3]
< 1 yr.	31 (129)[4]	55 (121)	46 (119)	9 (130)
1-5 yr.	6 (75)	20 (139)	16 (146)	4 (116)
> 5-15 yr.	4 (115)	8 (224*)[5]	3 (195)	5 (245*)
> 15 yr.	6 (105)	24 (220*)	5 (226)	19 (218*)
Over all	47 (114)	107 (144*)	70 (132*)	37 (175*)
Cumulative exposure (mppcf-year)[6] excluding employed < 3 months				
< 6	2 (68)	20 (106)	16 (101)	4 (131)
6-24	9 (112)	19 (131)	17 (154)	2 (59)
24-49	2 (55)	12 (200*)	5 (171)	7 (228*)
≥ 50	8 (101)	22 (206*)	4 (156)	18 (222*)
Dose-response relationship[7]		O/E = 1 + 0.0107x	O/E = 1 + 0.0100x	O/E = 1 + 0.0106x

NOTES:

1. Data is given for all workers in Plant II.
2. Data is given for workers in Plant II who were exposed to chrysotile only.
3. Data is given for workers in Plant II who were exposed to chrysotile and crocidolite.
4. Number in brackets is SMR.
5. Denotes statistic significant at $p<0.05$ (2-tailed test).
6. mppcf-year denotes mean million particle years per cubic foot.
7. O/E denotes observed to expected ratio; x denotes the level of exposure in mppcf-year.

TABLE 3

DOSE-RESPONSE RELATIONSHIP FOR LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS[1] FROM 1972-88

	Measures of Exposure Levels		
	Intensity	Duration of Employment[2]	Index
McDonald (1980) (Mining and Milling)	-	1-<5yr: 83 (29)[3] 5-<20yr: 137*[4] (50) ≥20yr: 161* (104)	<30mppcf.y: 93 (91) 30-<300mppcf.y: 118 (81) ≥300mppcf.y: 225* (70)
McDonald (1983a) (Pennsylvania) (Textiles)	-	1-<5yr: 33 (3) 5-<20yr: 129 (14) ≥20yr: 159* (27)	<10mppcf.y: 67 (21) 10-<20mppcf.y: 84 (5) 20-<40mppcf.y: 156 (10) 40-<80mppcf.y: 160 (6) ≥80mppcf.y: 416* (11)
McDonald (1983b) (South Carolina) (Textiles)	-	1-<5yr: 164 (10) 5-<20yr: 304* (15) ≥20yr.: 317* (26	<10mppcf.y: 143* (31) 10-<20mppcf.y: 183 (5) 20-<40mppcf.y: 304* (8) 40-<80mppcf.y: 420* (7) ≥80mppcf.y: 1032* (8)
Peto (1985) (Textiles)	1-199p/ml: 111 (27) 200-399p/ml: 193* (22) ≥400p/ml: 168* (36)	<10yr: 117 (53) ≥10yr: 209* (40) [20yr. +latency]	<1000p/ml.y: 115 (34) 1000p/ml.y: 104 (8) 2000p/ml.y: 167 (11) 3000p/ml.y: 106 (6) 4000p/ml.y: 233* (10) 5000+p/ml.y: 222* (24)

NOTES: Dose is expressed as:
intensity [i.e. fibres per cubic centimetre (f/cc), or particles per millilitre (p/ml), or simply as low, medium, heavy or severe];
duration [e.g. years of employment]; or as a
'dust index' [mppcf.y denotes mean million particle years per cubic foot; f/cc.y denotes fibre year per cubic centimetre; p/ml.y means particle years per millilitre];

1 Studies demonstrating both significant excess lung cancer deaths and 30 or more observed or expected lung cancer deaths are included. Latency for each study is as in Table 1 unless shown otherwise.

2 Duration of employment of less than one year is excluded.

3 SMR and observed lung cancer cases (in brackets) are presented for each exposure category.

4 * denotes a probability value $p < 0.05$.

TABLE 3 (CONT'D.)

DOSE-RESPONSE RELATIONSHIP FOR LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS FROM 1972-88

	Measures of Exposure Levels		
	Intensity	Duration of Employment	Index
Mcdonald (1984)	-	1-<5yr: 149 (19)	<10mppcf.y: 167 (55)
(Connecticut)		5-<20yr:123 (9)	10-<20mppcf.y: 102 (6)
(Friction Materials)		≥20yr: 133 (21)	20-<40mppcf.y: 105 (5)
			40-<80mppcf.y: 163 (6)
			≥80mppcf.y: 55 (1)
Hughes (1987)	-	1-5yr: 139 (20)	<6mppcf.yr: 106 (20)
(II)		>5-15yr:224* (8)	6-24mppcf.yr: 131 (19)
(Cement Products)		>15yr: 220* (24)	25-49mppcf.yr: 200*(12)
			50-99mppcf.yr: 181*(10)
			≥100mppcf.yr: 231*(12)
Raffn (1987)		1-4yr: 140 (11)	
(Incidence Study)		5+yr: 190* (59)	
(Cement Products)			
Acheson (1984)	low/casual:134 (12)	1-4yr: 308* (20)	-
(Insul.Products)	medium:225*(41)	5-9yr: 249* (8)	
	heavy:425* (8)	10-14yr:235* (6)	
		15+yr: 156 (7)	
		20+ yr.latency	
Seidman (1986)		1-2yr: 829* (16)	< 6f/cc.y: 288* (11)
(Insul.Products)		2+yr: 976* (24)	6-11.9f/cc.y: 649* (10)
		20+yr.latency	12-24.9f/cc.y: 628* (13)
			24-49.9f/cc.y: 464* (9)
			50-99.9f/cc.y: 696* (11)
			100-149.9f/cc.y:526* (5)
			150-249.9f/cc.y: 853*(7)
			250+f/ccc.y: 2273* (10)

NOTES: Dose is expressed as:
Intensity [i.e. fibres per cubic centimetre (f/cc), or particles per millilitre (p/ml), or simply as low, medium, heavy or severe];
Duration [e.g. years of employment]; or as a
'Dust Index' [mppcf.y denotes mean million particle years per cubic foot; f/cc.y denotes fibre years per cubic centimetre; p/ml.y means particle years per millilitre]. Studies demonstrating significant excess lung cancer deaths with 30 or more observed lung cancer deaths are included. Latency for each study is as in Table 1 unless shown otherwise. Risk estimates with duration of employment of less than one year are excluded. SMR and observed lung cancer cases (in brackets) are presented for each exposure category. * denotes a probability value p <0.05.

TABLE 3 (CONT'D.)

DOSE-RESPONSE RELATIONSHIP FOR LUNG CANCER MORTALITY IN COHORT STUDIES OF ASBESTOS WORKERS FROM 1972-88

	Measures of Exposure Levels		
	Intensity	Duration of Employment	Index
Enterline (1987) (General Mfg.)			<125mppcf.y: 182*(23) 125-249mppcf.y: 203*(14) 250-499mppcf.y: 322*(24) 500-749mppcf.y: 405*(10) ≥750mppcf.y: 699* (8)
Newhouse (1985) I (General Mfg.)	low or medium	<2yr: 150 (24)	-
	low or medium	>2yr: 180*(24)	
	severe	<2yr: 220*(43)	
	severe	>2yr: 480*(67)	
II	low or medium	250 (2)	
	severe	<2yr: 470*(14)	
	severe	>2yr:1750*(21)	
Hodgson (1986) (All Asbestos Work)	-	<10yr: 92 (40) ≥10yr: 141* (145)	-
Kolonel (1985) (Shipyard)	-	>15yr: 80 (27) ≥15yr: 140*(34)	-

NOTES: Dose is expressed as:
Intensity [i.e. fibres per cubic centimetre (f/cc), or particles per millilitre (p/ml), or simply as low, medium, heavy or severe];
Duration [e.g. years of employment]; or as a
'Dust Index' [mppcf.y denotes mean million particle years per cubic foot; f/cc.y denotes fibre years per cubic centimetre; p/ml.y means particle years per millilitre]. Studies demonstrating significant excess lung cancer deaths with 30 or more observed lung cancer deaths are included. Latency for each study is as in Table 1 unless shown otherwise. Risk estimates with duration of employment of less than one year are excluded. SMR and observed lung cancer cases (in brackets) are presented for each exposure category. * denotes a probability value $p < 0.05$.

TABLE 4

**INTERNATIONAL INCIDENCE RATES[1]
OF PLEURAL AND PERITONEAL MESOTHELIOMA**

PLEURAL MESOTHELIOMA

Reporting area (years)	Incidence[2]	M/F[3]	PL/PR[4]
Men:			
West Australia (1982)	2.8	-	20/2
Sweden (1978-82)	2.3	3.3	849/152
US Hawaii White (1978-82)	2.0	-	13/11
UK West Scotland (1978-82)	1.8	6.0	170/53
Switzerland, Basel (1981-82)	1.6	4.0	11/1
Canada (1978-82)	0.6	3.0	402/323
Ontario (1978-82)	0.4	4.0	109/66
Quebec (1978-81)	0.9	2.3	123/112
Women:			
Brazil, Porto Alegre (1979-82)	0.7	0.6	16/60
Sweden (1978-82)	0.7	3.3	320/118
India, Poona (1978-82)	0.6	1.5	12/3
Poland, Cracow City (1978-81)	0.6	0.8	12/13
Denmark (1978-82)	0.4	3.0	92/158
Canada (1978-82)	0.2	3.0	155/397
Ontario (1978-82)	0.1	4.0	40/59
Quebec (1978-81)	0.4	2.3	55/164

NOTES:

1 Reporting areas with the 5 highest incidences and reporting 10 or more cases: Canada, Ontario and Quebec rates are given. When rates of 2 or more areas are equal, the area with higher number of cases is given (Source IARC, 1987).

2 Number of cases per 100,000 per year, age adjusted to world population.

3 Ratio of male incidence to female incidence.

4 Ratio of number of pleural to number of peritoneal mesothelioma.

TABLE 4 (CONT'D.)

INTERNATIONAL INCIDENCE RATES[1]
OF PLEURAL AND PERITONEAL MESOTHELIOMA

PERITONEAL MESOTHELIOMA

Reporting area (years)	Incidence[2]	M/F[3]	PL/PR[4]
Men:			
Romania, County Cluj, Urban (1979-82)	2.8	1.6	2/25
Switzerland, Neuchatel (1978-82)	2.6	1.4	6/13
Brazil, Porto Alegre (1979-82)	1.8	0.6	5/26
Hawaii, White (1978-82)	1.6	5.3	13/11
Hungary, County Vas (1978-82)	1.3	2.6	5/12
Canada (1978-82)	0.5	1.0	402/323
Ontario (1978-82)	0.3	1.5	109/66
Quebec (1978-81)	0.8	0.7	123/112
Women:			
Brazil, Recife (1980)	4.2	0.4	7/19
Brazil, Porto Alegre (1979-82)	2.8	0.6	16/60
Romania, County Cluj, Rural (1978-82)	2.6	4.3	1/20
India, Bangolore (1982)	2.0	1.8	4/14
Switzerland, Neuchate (1978-82)	1.9	1.4	3/17
Canada (1978-82)	0.5	1.0	155/397
Ontario (1978-82)	0.2	1.5	40/59
Quebec (1978-81)	1.1	0.7	55/164

NOTES:

1 Reporting areas with the 5 highest incidences and reporting 10 or more cases; Canada, Ontario and Quebec rates are given. When rates of 2 or more areas are equal, the area with higher number of cases is given (Source IARC, 1987).

2 Number of cases per 100,000 per year, age adjusted to world population.

3 Ratio of male incidence to female incidence.

4 Ratio of number of pleural to number of peritoneal mesothelioma.

TABLE 5

MORTALITY FROM MESOTHELIOMA IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	MESOTHELIOMA DEATHS PERITONEAL	PLEURAL	CASE DESCRIPTIONS EXPOSURE[1]	LATENCY[2]
MINING AND MILLING:				
McDonald (1980)	0	10		20-55 yr
Rubino (1979)	0	1	33 yr. empl.	35 yr
Nicholson (1979)	0	1	40-49 yr.empl.	
Armstrong (1988)	1	32	<10 f/cc.y	>20 yr
Amandus (1987)	0	2	>300 f/cc.y	33 yr
TEXTILE:				
McDonald (1983) (Pennsylvania)	4	10		16-53 yr
McDonald (1983) (South Carolina)	1	0		42 yr
Dement (1983)	1	0		
Peto (1985) I:	0	7		
III:	0	11	>10 yr of employment	20-40+ yr
Robinson (1979) I:	5	4	(4 unspec.) 9-42 yr of employment	24-50 yr
II:	1	1	(2 unspec.)	

NOTES: [1] When the study indicates the exposure intensity, the values are indicated. Exposure is estimated by composite index (i.e. fibre concentration times duration of employment measured in fibre years per cubic centimetre or f/cc.y), or by duration of employment. Ranges or lower limits are given:

[2] Latency is defined as the elapsed time from initial exposure to death. Ranges or lower limits are given.

TABLE 5 (CONT'D.)

MORTALITY FROM MESOTHELIOMA IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	MESOTHELIOMA DEATHS PERITONEAL	PLEURAL	CASE DESCRIPTIONS EXPOSURE[1]	LATENCY[2]
GAS MASK MANUFACTURING:				
Acheson (1982) (Blackburn, Eng.)	0	1		
Jones (1980)	4	13	6-38 months of employment	
Acheson (1982) (Leyland, Eng.)	2	3		
Wignall (1982)	3	9		
McDonald (1978)	6	3	<2.5 yr of employment	
FRICTION MATERIAL:				
Berry (1983)	I: 0	8		
	II: 0	2		
McDonald (1984)	0	0		
Finkelstein (1988b)	0	1 (1 unspecified)		
CEMENT PRODUCTS:				
Hughes (1987)	I: 0	2	0.3 to 0.8 yr of employment	15-30 yr
	II: 1	8	0.4 to 42.3yr of employment	19-54 yr
Gardner (1986)	I: 0	1		7 yr
	II: 0	0		
Ohlson (1985)	0	0		

TABLE 5 (CONT'D.)

MORTALITY FROM MESOTHELIOMA IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	MESOTHELIOMA DEATHS PERITONEAL	PLEURAL	CASE DESCRIPTIONS EXPOSURE[1]	LATENCY[2]
Thomas (1982)	0	2		
Alies-Patin (1985)	1	3	14-27 yr of employment	
Finkelstein (1988a)	(31 unspecified)			10-30+ yr
Albin (1984)	0	11		
Raffn (1987)	1	12		
INSULATION PRODUCTS:				
Finkelstein (1987a)	0	0		
Finkelstein (1987b)	1	1		20-29 yr
Acheson (1984)	1	4		
Seidman (1986)	9	8		
GENERAL MANUFACTURING:				
Weiss (1977)	0	2		
Enterline (1987)	2 (1 unspecified)	5	48-760 f/cc.y 29-55 yr of employment	
Newhouse I: (male) (1985)	29	31	low to severe exposure <2yr of employment	
II: (female)	11	14	mainly severe exposure <2yr of employment	
INSULATION:				
Selikoff (1979) (Canada & USA)	112	63	170 cases >20 yr	

TABLE 5 (CONT'D.)

MORTALITY FROM MESOTHELIOMA IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS		MESOTHELIOMA DEATHS PERITONEAL	PLEURAL	CASE DESCRIPTIONS EXPOSURE[1]	LATENCY[2]
Robinson (1979)	I:	5	4 (4 unspec.)	9-42 yr	24-50 yr
Newhouse (1985)		6	7		
Elmes (1977)		5	8		
ALL ASBESTOS WORK:					
Hodgson (1986)	I:	34 (unspec.)			
	II:	1 (unspec.)			
Woltowitz (1986)	I:	0	6		
	II:	2	4		
SHIPYARD:					
Kolonel (1985)		0	7	1-29 yr.	13-41 yrs
ELECTRO-CHEMICAL:					
Hilt (1985)	I:	1	4		
SHEET METAL:					
Michaels (1988)		0	3 (3 unspecified)		
PLUMBING AND PIPEFITTING:					
Cantor (1986)		1	12 (3 unspecified)		
RAILROAD WORK:					
Ohlson (1984)		(5 unspecified)			30-57 yrs
Mancuso (1988)		0	13 (1 unspecified)		33-64 yrs

TABLE 6

MORTALITY FROM CANCERS OF THE LARYNX, ESOPHAGUS, STOMACH, COLON AND RECTUM IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	NUMBER OF DEATHS FROM CANCER OF[1]:				
	LARYNX	ESOPHAGUS	STOMACH	COLON & RECTUM	GI TRACT
MINING AND MILLING:					
McDonald (1980)	16 (107)[2]	130 (eso. & stom.) (127)		79 (78)	-
Rubino (1979)	6 (316*)[4]	-	-	-	19+[3] (98)
Nicholson (1979)	-	-	-	-	10 (105)
Armstrong (1988)	2 (109)	3 (118)	17 (190*)	14 (114)	-
Amandus (1987)	-	-	2 (124)	-	6+ (74)
McDonald (1986)					7 (88)

NOTES: [1] Groupings by site are based on the following ICD codes:

Larynx	ICD 161
Esophagus	ICD 150
Stomach	ICD 151
Colon & Rectum	ICD 153, 154
GI Tract	ICD 150-154

[2] SMR shown in brackets below the observed number of cases;

[3] + denotes ICD 150-159 grouping

[4] * indicates a significant SMR at the 5% level.

TABLE 6 (CONT'D.)

MORTALITY FROM CANCERS OF THE LARYNX, ESOPHAGUS, STOMACH, COLON AND RECTUM IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	NUMBER OF DEATHS FROM CANCER OF[1]:				
	LARYNX	ESOPHAGUS	STOMACH	COLON & RECTUM	GI TRACT
TEXTILE:					
McDonald (1983a)	0	-	-	-	54[+,§] (113)
McDonald (1983b)	0	-	-	-	26 (152*)
Dement (1983)	-	-	-	-	13[+] (131)
Peto (1985) I:	0	0	4 (176)	3 (145)	-
II:	0	0	2 (185)	4 (198)	-
III:	4 (155)	11 (177)	29 (100)	20 (75)	-
Robinson (1979) I:	-	-	-	-	50 (121)
II:	-	-	-	-	8 (133)
GAS MASK MANUFACTURING:					
Acheson (1982)	-	-	4 (129)	-	-
Jones (1980)	-	-	-	-	10 (49*)
Acheson (1982) (Leyland, Eng.)	-	-	5 (114)	-	-
Wignall (1982)	-	-	-	-	7 (65)

NOTE: [§] [**] denotes the grouping was reported as abdominal cancer

TABLE 6 (CONT'D.)

MORTALITY FROM CANCERS OF THE LARYNX, ESOPHAGUS, STOMACH, COLON AND RECTUM IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	NUMBER OF DEATHS FROM CANCER OF[1]:				
	LARYNX	ESOPHAGUS	STOMACH	COLON & RECTUM	GI TRACT
FRICTION MATERIAL:					
Berry (1983) I:	2 (55)	-	-	-	103* (96)
II:	0	-	-	-	29 (106)
McDonald (1984)	-	-	-	-	59 (114)*
Finkelstein (1988b)	4 (667*)	-	-	-	9 (87)*
CEMENT PRODUCTS:					
Hughes (1987) I:	2 (100)	5 (100)	8 (107)	10 (120)	-
II:	1 (30)	7 (89)	15 (125)	11 (73)	-
Gardner (1986)	1 (90)	1 (29)	15 (109)	11 (71)	-
Ohlson (1985)	-	-	1 (17)	11 (186)	-
Thomas (1982)	-	-	-	-	18 (92)
Finklestein (1988a)	0	-	-	-	17 (86)
Albin (1984)	-	-	-	-	19 (176*)
Raffn (1987) (Incidence Study)	14 (165)	-	43 (143)*	67 (116)	143 (118*)

TABLE 6 (CONT'D.)

MORTALITY FROM CANCERS OF THE LARYNX, ESOPHAGUS, STOMACH, COLON AND RECTUM IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	NUMBER OF DEATHS FROM CANCER OF[1]:				
	LARYNX	ESOPHAGUS	STOMACH	COLON & RECTUM	GI TRACT
INSULATION PRODUCTS:					
Finkelstein (1987a)	0	-	-	-	3 (120)
Finkelstein (1987b)	0	-	-	-	0
Acheson (1984)	-	2 (100)	7 (94)	10 (132)	-
Seidman (1986)	7[#6] (192)	1 (49)	11 (190*)	22 (185*)	-
GENERAL MANUFACTURING:					
Weiss (1977)	-	-	-	-	14 (99)
Enterline (1987)	2 (114)	4 (136)	20 (180)*	23 (116)	-
Newhouse (1985) I:	-	-	-	-	67 (133)*
II:	-	-	-	-	23 (182*)*

NOTE: [6] [#] denotes larynx, buccal and pharynx cancer.

TABLE 6 (CONT'D.)

MORTALITY FROM CANCERS OF THE LARYNX, ESOPHAGUS, STOMACH, COLON AND RECTUM IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS	NUMBER OF DEATHS FROM CANCER OF[1]: LARYNX	ESOPHAGUS	STOMACH	COLON & RECTUM	GI TRACT
INSULATION:					
Selikoff (1979) (Canada & USA)	11 (234*)	18 (254*)	22 (155*)	59 (154*)	-
Selikoff (1979) (New York, New Jersey)	6[2] (214)	1 (71)	19 (352*)	23 (277*)	-
Newhouse (1985)	-	-	-	-	11 (136)*
Elmes (1977)	-	-	-	-	13 (1300*)
ALL ASBESTOS WORK:					
Hodgson (1986) I:	-	6 (64)	27 (100)	16 (54*)	-
II:	-	1 (44)	5 (83)	4 (55)	
Woitowitz (1986) I:	-	13 (eso. & stom.) (182*)		5 (79)	-
II:	-	2 (eso. & stom.) (142)		3 (215)	-
SHIPYARD:					
Kolonel (1985)	-	-	-	-	45 (96)
Rossiter (1980)	-	-	-	-	63 (83)

TABLE 6 (CONT'D.)

MORTALITY FROM CANCERS OF THE LARYNX, ESOPHAGUS, STOMACH, COLON AND RECTUM IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

INVESTIGATORS		NUMBER OF DEATHS FROM CANCER OF[1]: LARYNX	ESOPHAGUS	STOMACH	COLON & RECTUM	GI TRACT
ELECTRO-CHEMICAL:						
Hilt (1985)	I:	-	0	0	2 (140)	-
	II :	-	0	2 (125)	3 (390)	-
SHEET METAL:						
Michaels (1988)		1 (97)[p&]	1 (61)[p]	7 (259*)[p]	12 (132)[p]	-
PLUMBING AND PIPEFITTING:						
Cantor (1986)		25 (117)[p]	35 (106)[p]	93 (129*)[p]	165 (94)[p]	-
RAILROAD WORK:						
Ohlson (1984)		-	41 (eso. & stom.) (57)		20 (66)	-

NOTE: [1] [p] denotes that Proportional Mortality Ratios (PMRs) are shown.

TABLE 7

RELATIONSHIP OF LATENCY TO THE MORTALITY EXPERIENCE FROM GASTROINTESTINAL CANCERS IN COHORT STUDIES OF ASBESTOS WORKERS REPORTED FROM 1972-88

Cohort	Latency Period	GI Deaths Obs	SMR
Selikoff (1979)	< 20 yr	6	97
	>= 20 yr	93	175*
Finkelstein (1988a)'	overall	14	113*
	>= 20 yr	14	194*
Albin (1984)	overall	19	176*
	>= 20 yr	17	213*
Peto (1985)	< 20 yr	20	82
	>= 20 yr	40	106
Seidman (1986)	5-19 yr	12	125
	5-39 yr	34	172*
	20-39 yr	22	216*

NOTE: * denotes a statistically significant SMR at the 5% level.

' denotes that the indicated results are restricted to workers with > 1 year of employment.

TABLE 8

DESIGN OF COHORT AND CASE-CONTROL STUDIES
OF 4 ONTARIO ASBESTOS PLANTS

	DOMTAR[1]	P[illegible]ES[2]	JOHNS-MANVILLE[3]	BENDIX[4]
Location	Cornwall	Sarnia	West Hill	Windsor
Product (fibre type)	fibre conduit insulation boards (chrysotile)	pipe insulation products (amosite)	rock wool insulation (chrysotile & crocidolite) cement board (chrysotile) insulation material (amosite)	brake shoes (chrysotile)
Period of operation	1929-82	1956-74	1947-80	1929-80
Dust/fibre level	never measured	1958: 2.1-134.4mppcf 1973: 1-852 f/cc	1949: 8-40 f/cc[5] 1969: 4-20 f/cc 1979: .3-0.5 f/cc	< 2f/cc (1975 on)
Follow-up study: number in exposed cohort[6]	324	107	3,166	1,657 (343 females)
Period of follow-up	1960-85	1956-87	1950-85	1950-85
Inclusion criterion	hired before Jan.1 1974	all exposed workers	all exposed workers	> 1 yr employed
Duration of employment	21% < 1 yr 40% > 10 yr	50% < 2 yr	50% < 1 yr 12% ≥10 yr	45% < 5 yr 38% >10 yr

NOTES:
1 Finkelstein 1987a
2 Finkelstein 1987b
3 Finkelstein 1988a
4 Finkelstein 1988b
5 Finkelstein 1984
6 All cohorts are male workers except for Bendix.

TABLE 8

DESIGN OF COHORT AND CASE-CONTROL STUDIES OF 4 ONTARIO ASBESTOS PLANTS

	DOMTAR[1]	HOLMES[2]	JOHNS-MANVILLE[3]	BENDIX[4]
Number of deaths from all causes (SMR)	51 (93)	13 (142)	408 (104)	186 (m) (105) 32 (f) (178*)
CASE-CONTROL STUDY:				
case control ratio	1:3	-	1:3	1:3
matching factors	age	-	yr of birth yr of hire	yr of birth gender

NOTE: * denotes a statistically significant SMR.

TABLE 9

LUNG CANCER MORTALITY DATA FROM STUDIES OF ASBESTOS WORKERS IN 4 ONTARIO MANUFACTURING PLANTS

	DOMTAR[1]	BOLMES[2]	JOHNS-MANVILLE[3]	BENDIX[4]
COHORT STUDY:				
Number of lung cancer deaths in exposed cohort (SMR)	6 (162)	4 (606*)	49 (168*)	15 (124)
Critical duration of employment[5]	>=10 yr		>1 yr	nc[6]
Critical latency[7]	>=20 yr		>=20 yr	nc
Number of deaths (SMR)	6 (220*)		31 (265*)	

NOTES:
1 Finkelstein 1987a
2 Finkelstein 1987b
3 Finkelstein 1988a
4 Finkelstein 1988b
5 Critical duration of employment is that duration at which a significant excess of deaths is observed.
6 nc denotes no critical level was either detected or available.
7 Critical latency period is that latency at which a significant excess of deaths is observed.
* denotes a statistically significant result.

TABLE 9 (CONT'D.)

LUNG CANCER MORTALITY DATA FROM STUDIES OF ASBESTOS WORKERS IN 4 ONTARIO MANUFACTURING PLANTS

	DOMTAR[1]	HOLMES[2]	JOHNS-MANVILLE[3]	BENDIX[4]
CASE-CONTROL STUDY:				
Critical duration of employment	-	-	1-10 yr	nc
Odds ratio [confidence interval]			1.8 [0.9,3.6]	
Trend with duration of employment	-	-	$p<0.001$	-
Highest risk	beater-operator	-	>=10 yr employment	-
Odds ratio [conf.interval]	7.2 [2.3, 23.3][1]	-	4.5 [2.4, 8.7]	-

NOTES: [1] 95% confidence intervals for the Domtar study, and 90% conf. int. for Johns-Manville study.

TABLE 10

MORTALITY EXPERIENCE FROM CANCERS OF THE LARYNX, GASTROINTESTINAL TRACT AND MESOTHELIOMA AMONG ASBESTOS WORKERS IN FOUR ONTARIO MANUFACTURING PLANTS

	DOMTAR[1]	HOLMES[2]	JOHNS-MANVILLE[3]	BENDIX[4]
LARYNGEAL CANCER:				
1. Number of deaths in entire exposed cohort (SMR)	0	0	0	4[#5] (667*)[6]
GASTROINTESTINAL CANCER:				
1. Number of deaths in entire exposed cohort (SMR)	3 (120)	0	17 (86)	9 (87)
2. Critical duration of employment and latency:	nc		>1 yr & >=20 yr	nc
3. Number of deaths (SMR)			14 (194*)	
MESOTHELIOMA:				
1. Number of deaths in entire exposed cohort	0	2	31	2
2. Latency range		20-29 yr	10-30+ yr	10-19 yr

NOTES:
1 Finkelstein 1987a
2 Finkelstein 1987b
3 Finkelstein 1988a
4 Finkelstein 1988b
5 # denotes that all 4 men were employed more than 10 years and that all worked in non-asbestos using area.
6 * denotes a significant statistic at the 5% level.

BIBLIOGRAPHY

Acheson,E.D.: Gardner, M.J.: et al. Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40 year follow-up. British journal of industrial medicine. Vol.39(1982). p.344-348.

Acheson,E.D.:Gardner, M.J. Cancer in a factory using amosite asbestos International journal of epidemiology. Vol.13(1984). p.3-10.

Albin, M., Jakobsson, K., Englander, V. et al. Mortality and cancer morbidity in a cohort of asbestos cement workers. In: VIth international pneumoconiosis conference 1983: v.2; September, 1983; Bochum, West Germany. Bremerhaven, West Germany: Wirtschaftsverlag NW, Verlag fur neue Wissenschaft GmbH.p. 825-829.

Alies-Patin, A.M;Valleron A.J. Mortality of workers in a French asbestos cement factory 1940-82. British journal of industrial medicine. Vol.42(1985). p.219-225.

Amandus, H.E.: Wheeler, R. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite: part II. mortality. American journal of industrial medicine. Vol.11(1987). p.15-26.

Armstrong,B.K.: De Klerk,N.H.: et al. Mortality in miners and millers of crocidolite in Western Australia. British journal of industrial medicine. Vol. 45(1988). p.5-13.

Becklake, MR. On Dr. Dunnigan's commentary linking chrysotile asbestos with mesothelioma. American journal of industrial medicine. Vol.14(1988). p.239-240. (letter to the editor)

Benhamou, S.: Benhamou, E.: Flamant, R. Occupational risk factors of lung cancer in a French case-control study. British journal of industrial medicine. Vol.45(1988). p.231-233.

Berry, G.: Newhouse, M.L.: Turok, M. Combined effect of asbestos exposure and smoking on mortality from lung cancer in factory workers. The Lancet. Sept. 2, 1972. p.476-479.

Berry, G.: Newhouse,M.L. Mortality of workers manufacturing friction materials using asbestos. British journal of industrial medicine. Vol.40(1983). p.1-7.

Berry: G: Newhouse,M.L.: Antonis, P. Combined effect of asbestos and smoking on mortality from lung cancer and mesothelioma in factory workers. British journal of industrial medicine. Vol.42(1985). p.12-18.

Brown, LM: Mason, TJ: Pickle, LW: et al. Occupational risk factors for laryngeal cancer on the Texas Gulf Coast. Cancer research. Vol.48(1988). p.1960-1964.

Burch, J.D.: Howe, G.R.:Miller,A.B. Tobacco, alcohol, asbestos and nickel in the etiology of cancer of the larynx: a case-control study. Journal of the National Cancer Institute. Vol.67(1981). p.1219-1224.

Cantor, K.P: Sontag, J.M.:Held, M. Patterns of mortality among plumbers and pipefitters. American journal of industrial medicine. Vol.10(1986). p.73-89.

Churg, A; Wiggs, B. Fibre size and number in amphibole asbestos-induced mesothelioma. American journal of pathology. Vol.115(1984). p.437-442.

Churg, A. Chrysotile, Tremolite, and malignant mesothelioma in man. Chest. Vol.93(1986). p.621-628.

Churg, A. Reply to Dr. Dunnigan (letter to the editor). American journal of industrial medicine. Vol.14(1988). p.235-238.

Clarke, E.A.; Kreiger, N. and Marrett, L.D. Cancer Incidence, Mortality and Treatment in Ontario. In: Cancer in Ontario, 1985. The Ontario Cancer Treatment and Research Foundation. 1985.

Craighead, JM. Response to Dr. Dunnigan's commentary. American journal of industrial medicine. Vol.14(1988). p.241-243. (letter to the editor)

Damber, L.A.; Larsson, L.G. Occupation and male lung cancer: a case-control study in northern Sweden. British journal of industrial medicine. Vol.44(1987). p.446-453.

Davis, JMG. On the statement by Jacques Dunnigan. American journal of industrial medicine. Vol.14(1988). p.629-630. (Commentary)

Dement,J.M.; Harris,R.L. Exposures and mortality among chrysotile asbestos workers. Part II: mortality. American journal of industrial medicine. Vol.4(1983). p.421-423.

Doll, R. and Peto, J. Effect on health of exposure to asbestos. Health and Safety Commission (1985). London: Her Majesty's Stationery Office.

Dunnigan, J. Linking chrysotile asbestos with mesothelioma (commentary). American journal of industrial medicine. Vol.14(1988). p.205-209.

Edelman, D.A. Exposure to asbestos and the risk of gastrointestinal cancer: a reassessment. British journal of industrial medicine. Vol.45(1988). p.75-82.

Elmes, P.C.;Simpson, M.J.C. Insulation workers in Belfast. A further study of mortality due to asbestos exposure (1949-75). British journal of industrial medicine. Vol.34(1977). p.174-180.

Elwood, J.M.; Pearson, J.C.G.; Skipper, D.H. Alcohol, smoking, social and occupational factors in the aetiology of cancer of the oral cavity, pharynx and larynx. International journal of cancer. Vol.34(1984). p.603-612.

Enterline, P.E. Multiple causal factors in individual cases. In: Methods and issues in occupational and environmental epidemiology. Ann Arbor: Ann Arbor Science, 1983. p.177-182.

Enterline, P.E.; Hartley,J.;Henderson,V. Asbestos and cancer: a cohort followed up to death. British journal of industrial medicine. Vol.44, no.6(1987). p. 396-401.

Finkelstein, M.M.; Kusiak R.A. A study of mortality among employees of the Bendix Automotive Corporation, Windsor, Ontario. Toronto: Ontario Ministry of Labour, 1983.

Finkelstein,M.M. Mortality among employees of an Ontario asbestos-cement factory. American review of respiratory disease. Vol.129(1984). p.754-761.

Finkelstein, M.M. Mortality among former employees of the Domtar pipe factory in Cornwall, Ontario. Ontario Ministry of Labour. Toronto. 1987a.

Finkelstein, M.M. Mortality among employees of a Sarnia Ontario factory which manufactured insulation materials from amosite asbestos. Ontario Ministry of Labour. 1987b.

Finkelstein, M.M. Cancer among former employees of the Johns-Manville asbestos-cement factory in West Hill, Ontario: An updated analysis and assessment of risk. Ontario Ministry of Labour. Toronto. 1988a.

Finkelstein, M.M. Mortality among employees of the Bendix Automotive Corporation, Windsor, Ontario. Ontario Ministry of Labour. Toronto. 1988b.

Frumkin, H.; Berlin, J. Asbestos exposure and gastrointestinal malignancy, review and meta-analysis. American journal of industrial medicine. Vol.14(1988). p.79-95.

Gardner, M.J.; Winter, P.D.; et al. Follow up study of workers manufacturing chrysotile asbestos cement products. British journal of industrial medicine. Vol.43(1986). p.726-732.

Gardner, M.J.; Powell, C.A.; et al. Continuing high lung cancer mortality among ex-amosite asbestos factory workers and a pilot study of individual anti-smoking advice. Journal of the Society for Occupational Medicine. Vol 38(1988). p.69-72.

Hammond, E.C.;Selikoff,I.J.; Seidman, H. Asbestos exposure, cigarette smoking and death rates. Annals of the New York Academy of Sciences. Vol. 330(1979). p.473-490.

Hilt, B.; Langard, S; et al. Asbestos exposure, smoking habits, and cancer incidence among production and maintenance workers in an electrochemical plant. American journal of industrial medicine. Vol.8(1985). p.565-577.

Hinds, M.W., Thomas, D.B., O'Reilly, H.P.: Asbestos, dental x-rays, tobacco and alcohol in the epidemiology of laryngeal cancer. Cancer. Vol.44(1979). p.1144-1120.

Hodgson,J.T.; Jones,R.D. Mortality of asbestos workers in England and Wales. British journal of industrial medicine. Vol.43(1986). p.158-164.

Hughes, J.M.; Weill, H.; Hammad,Y. Mortality of workers employed in two asbestos cement manufacturing plants. British journal of industrial medicine. Vol.44(1987). p.161-174.

Jones, J.S.; Smith, P.G.; et al. The consequences of exposure to asbestos dust in a wartime gas-mask factory. In: Wagner, JC, Davis, W, eds. Biological effects of mineral fibres. (IARC publication no. 30). Lyon: International Agency for Research on Cancer, 1979. p.637-653.

Kogan, FM: Re: Chrysotile asbestos and mesothelioma of pleura. American journal of industrial medicine. Vol.15(1988). p.123. (letter to the editor)

Kolonel, L.N.; Yoshizawa, C.N.; et al. Cancer occurrence in shipyard workers exposed to asbestos in Hawaii. Cancer research. Vol.45(1985). p.3924-3928.

Levine, D.S. Does asbestos exposure cause gastrointestinal cancer? Digestive diseases and science. Vol.30(1985), no. 12. p.1189-1198.

Liddell FDK, Thomas DC, Gibbs GW and McDonald JC: Fibre exposure and mortality from pneumoconiosis, respiratory and abdominal Malignancies in chrysotile production in Quebec, 1926-75. Singapore Annals Academy of Medicine. Vol.13, no.2(1984),supp. p.340-344.

Lilienfeld, DE. Obfuscation: a reply to Dr. Dunnigan. American journal of industrial medicine. Vol.14(1988). p.625-628. (Commentary)

Lynch, H.T.; Katz, D., et al. Familial mesothelioma: review and family study. Cancer genetics and cytogenetics. Vol.15,no.1-2(1985). p.121-126.

Malker, H.S.; McLaughlin, J.K. Peritoneal mesothelioma in the construction industry in Sweden. Journal of occupational medicine. Vol.29,no.12(1987). p.979-980.

Mancuso, T.F. Relative risk of mesothelioma among railraod machinists exposed to chrysotile. American journal of industrial medicine. Vol.13(1988) p.639-657.

McDonald, A.D.; McDonald, J.C. Mesothelioma after crocidolite exposure during gas mask manufacture. Environmental research. Vol. 17(1978). p.340-346.

McDonald, J.C.; Liddell, F.D.K.; et al. Dust exposure and mortality in chrysotile mining, 1910-1975. British journal of industrial medicine. Vol.37(1980). p.11-24.

McDonald, A.D.; Fry, J.S.; et al. Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. British journal of industrial medicine. Vol.39(1983a)
p.368-374.

McDonald, A.D.; Fry, J.S.; et al. Dust exposure and mortality in an American chrysotile textile plant. British journal of industrial medicine. Vol.40(1983b). p.361-367.

McDonald, A.D.; Fry,J.S.;et al. Dust exposure and mortality in an American chrysotile asbestos friction products plant. British journal of industrial medicine. Vol.41(1984). p.151-157.

McDonald, JC; McDonald, AD; Armstrong, B; Sebestien, P. Cohort study of mortality of vermiculite miners exposed to tremolite. British journal of industrial medicine. Vol.43(1986). p.436-444.

McDonald, AD; McDonald, JC. Epidemiology of malignant mesothelioma. In: Asbestos related malignancy. Grune and Stratton, Inc. 1986.

McDonald, JC. Tremolite, other amphiboles and mesothelioma. American journal of industrial medicine. Vol.14(1988). p.247-249. (letter to the editor)

Michaels, D.; Zoloth, S. Asbestos disease in sheet metal workers: proportional mortality update. American journal of industrial medicine. Vol.13(1988). p.731-734.

Miller, A.B. Asbestos fibre dust and gastro-intestinal malignancies: Review of literature with regard to a cause/effect relationship. Journal of chronic disease. Vol.31 (1978). p.23-33.

Monson R.R. Occupational Epidemiology. CRC Press Inc. Boca Raton, Florida. 1987.

Morgan, R.W.; Foliart, D.E.; Wong, O. Asbestos and gastrointestinal cancer, a review of the literature. The Western journal of medicine. Vol.143(1985). p.60-65.

Muir, C; Waterhouse, J; et al. eds. Cancer incidence in five continents. Vol.V. International Agency for Research on Cancer, Lyon, 1987.

Newhouse,M.L.; Berry,G.; Wagner, J.C. Mortality of factory workers in east London 1933-80. British journal of industrial medicine. Vol.42(1985). p.4-11.

Nicholson, W.J. Case study 1: Asbestos- the TLV approach. Annals of the New York Academy of Sciences. Vol.271(1976). p.152-169.

Nicholson, W.J.; Selikoff,I.J.;et al. Long-term mortality experience of chrysotile miners and millers in Thetford Mines, Quebec. Annals of the New York Academy of Sciences. Vol.330(1979) p.11-21.

Nicholson, W.J. Airborne asbestos health assessment update.
The Environmental Criteria and Assessment Office, US Environmental Protection Agency,1986.

Ohlson, C.G.; Klaesson,B. Mortality among asbestos exposed workers in a railroad workshop. Scandinavian journal of work, environment, and health. Vol.10(1984). p.283-291.

Ohlson,C.G.; Hogstedt,C. Lung cancer among asbestos cement workers. A Swedish cohort study and a review. British journal of industrial medicine. Vol. 42(1985). p.397-402.

Olsen, J.; Sabroe, S. Occupational causes of laryngeal cancer. Journal of epidemiology and community health. Vol.38(1984). p.117-121.

Peto, J.; Doll, R.; et al. Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. Annals of occupational hygiene. Vol.29,no.3(1985). p. 305-355.

Pelnar, P. Further evidence of nonasbestos-related mesothelioma, a review of the literature. Scandinavian journal of work and environmental health. Vol.14(1988). p.141-144.

Raffn E , Lynge E., Juel K and Korsgaard B: Cancer incidence and mortality among employees at an asbestos-cement factory in Denmark. Cancerregisteret. Institut for Cancer Epidemiologi under Kraeftens Bekaempelse. 1987.

Report of The Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. Ontario Ministry of the Attorney General. 3 vols. 1984.

Robinson, C.; Lemen, R.; et al. Mortality patterns, 1940-1975, among workers employed in an asbestos textile friction and packing products manufacturing facility. In: Lemen, R, Dement, JR, eds. Dusts and disease: proceedings of the conference on occupational exposures to fibrous and particulate dust and their extension into the environment. Forest Park, Il: Pathotox Publishers, 1979. p.131-143.

Roggli, VL. Amphiboles and chrysotile exposure. American journal of industrial medicine. Vol.14(1988). p.245-246. (letter to the editor)

Rossiter, C.E.;Coles, R.M. HM Dockyard, Devonport: 1947 mortality study. In: Wagner, JC, Davis, W, eds. Biological effects of mineral fibres. (IARC scientific publication no. 30). Lyon: International Agency for Research in Cancer, 1980. Vol.2. p.713-721.

Rubino, G.F.; Piolatto, G. Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. British journal of industrial medicine. Vol.36(1979). p.187-194.

Schenker, M.B.; Garshick, R.A. A population-based case-control study of mesothelioma deaths among U.S. railroad workers. American review of respiratory disease. Vol.134(1986). p.461-465.

Schoenberg,J.B.; Stemhagen, A.; et al. Occupation and lung cancer risk among New Jersey white males. Journal of the National Cancer Institute. Vol.79, no.1(1987). p.13-21.

Seidman, H.; Selikoff,I.J.;Gelb,S.K. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposure. American journal of industrial medicine. Vol.10(1986). p.479-514.

Selikoff, I.J.; Hammond, E.C.; Seidman, H. Mortality experience of insulation workers in the United States and Canada, 1943-1976. Annals of the New York Academy of Sciences. Vol.330(1979). p.91-116.

Sluis-Cremer, GK. Linking chrysotile asbestos with mesothelioma. American journal of industrial medicine. Vol.14(1988). p.631-632. (letter to the editor)

Thomas, H.F.; Benjamin,I.T.; et al. Further follow-up study of workers from an asbestos cement factory. British journal of industrial medicine. Vol. 39(1982). p.273-276.

Weiss, W. Mortality of a cohort exposed to chrysotile asbestos. Journal of occupational medicine. Vol.19,no.11(1977) p.737-740.

Wignall,B.K.; Fox, A.J. Mortality of female gas mask assemblers. British journal of industrial medicine. Vol.39(1982). p.34-38.

Woitowitz,H.J.; Lange,H.J.; et al. Mortality rates in the Federal Republic of Germany following previous occupational exposure to asbestos dust.
International archives of occupational and environmental health. Vol.57(1986). p.161-171.

APPENDIX B

EVIDENTIARY SOURCE INFORMATION

ON

ASBESTOS-RELATED CANCERS

APPENDIX B

EVIDENTIARY SOURCE INFORMATION ON ASBESTOS-RELATED CANCERS

Abratt,R.P.; Willcox,P.A. Mesothelioma - short latent period after industrial asbestos exposure and prolonged survival. European journal of surgical oncology. Vol.11(1985). p.279-282.

Acheson,E.D.; Gardner,M.J.; et al. Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40 year follow up. British journal of industrial medicine. Vol.39(1982). p.344-348.

Acheson,E.D.; Gardner,M.J.; et al. Cancer in a factory using amosite asbestos. International journal of epidemiology. Vol.13(1984). p.3-10.

Albin,M.; Jakobsson,K.; et al. Mortality and cancer morbidity in a cohort of asbestos-cement workers. In : VIth international pneumoconiosis conference 1983:vol.2; Bochum, West Germany. Bremerhaven, West Germany: Wirtschaftsverlag NW, Verlag fur neue Wissenschaft GmbH., 1983. p.825-829.

Amandus,H.E.; Wheeler,R.; et al. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite: part 1-exposure estimates. part2-mortality. part3-radiographic findings. American journal of industrial medicine. Vol.11(1987). p.1-37.

Anderson,H.A.; Selikoff,I.J. Asbestos-associated radigraphic changes among household contacts of amosite asbestos workers. In: Preger,L.,ed. Induced disease: drug, irradiation, occupation. Orlando,Fl.: Grune and Stratton, 1979. p.253-273.

Anderson,H.A. Family contact exposure. In : Proceedings of the World Symposium on Asbestos. (1983). p.349-362.

Anderson,H.A.; Lilis,R.; et al. Household-contact asbestos neoplastic risk. Annals of the New York Academy of Sciences. Vol.271(1976). p.311-323.

Anderson,H.; Lilis,R.; et al. Household exposure to asbestos and risk of subsequent disease. In: Lemen,R.:Dement,J.M.,eds. Dusts and disease. Park Forest, Ill.: Pathotox, 1979. p.145-156.

Anderson,H.A.; Lilis,R.; et al. Asbestosis among household contacts of asbestos factory workers. Annals of the New York Academy of Sciences. Vol.330(1979). p.387-399.

Armstrong,B.K.; Klerk,N.H.; et al. Mortality in miners and millers of crocidolite in Western Australia. British journal of industrial medicine. Vol.45(1988). p.5-13.

Ashcroft,T.; Heppleston,G. Mesothelioma and asbestos on Tyneside - a pathological and social study. In : Pneumoconiosis - Proceedings of the International Conference, Johannesburg 1969. Cape Town : Oxford University Press, 1970. p.177-179.

Barnes,R. Compensable asbestos-related disease in New South Wales. The medical journal of Australia. Vol.2(1983). p.221-224.

Barth,P.S. Workers' compensation and asbestos in Ontario. [Report prepared for the Royal Commission on Matters of Health and Safety Arising from the use of Asbestos in Ontario]. Study no.2. January, 1982.

Barth,P.S. [Response of 1982 09 02 to critique of Workers' Compensation and Asbestos in Ontario by W.J. McCracken of the WCB's Medical Services Division].

Barth,P.S. Compensation for asbestos-associated disease : a survey of asbestos insulation workers in the United States and Canada. Report to the U.S. Department of Labor Assistant Secretary for Policy, Evaluation and Research. January, 1981.

Becklake,M.R. On Dr. Dunnigan's commentary linking chrysotile asbestos with mesothelioma. [Letter to the Editor]. American journal of industrial medicine. Vol.14, no.2(1988). p.239-240.

Becklake,M.R. Asbestos-related diseases of the lung and other organs: their epidemiology and implications for clinical practice. American review of respiratory disease. Vol.114(1976). p.187-227.

Benhamou,S.: Benhamou,E.: Flamant,R. Occupational risk factors of lung cancer in a French case-control study. British journal of industrial medicine. Vol.45(1988). p.231-233.

Berry,G.: Newhouse,M.L. Mortality of workers manufacturing friction materials using asbestos. British journal of industrial medicine. Vol.40(1983). p.1-7.

Berry,G.: Newhouse,M.L.: Antonis,P. Combined effect of asbestos and smoking on mortality from lung cancer and mesothelioma in factory workers. British journal of industrial medicine. Vol.42(1985). p.12-18.

Berry,G.: Newhouse,M.L.: Turok,M. Combined effect of asbestos exposure and smoking on mortality from lung cancer in factory workers. The Lancet. (Sept.2, 1972). p.476-479.

Britton,M. Compensation for asbestos-related diseases - the U.K. model. Respiratory medicine. Vol.83(1989). p.95-102.

Browne,K. A threshold for asbestos related lung cancer. British journal of industrial medicine. Vol.43(1986). p.556-558.

Brown,L.M.: Mason,T.J.: et al. Occupational risk factors for laryngeal cancer on the Texas gulf coast. Cancer research. Vol.48(April 1, 1988). p.1960-64.

Browne,K. The epidemiology of mesothelioma. Journal of the Society of Occupational Medicine. Vol.33(1983). p.190-194.

Browne,K. Is asbestos or asbestosis the cause of the increased risk of lung cancer in asbestos workers? British journal of industrial medicine. Vol.43(1986). p.145-149.

Bruckman,L.: Rubino,R.A.: Christine,B. Asbestos and mesothelioma incidence in Connecticut. Journal of the Air Pollution Control Association. Vol.27, no.2(Feb. 1977). p.121-126.

Champion,P. Two cases of malignant mesothelioma after exposure to asbestos. American review of respiratory disease. Vol.103(1971). p.821-826.

Chan,C.K.; Gee,B.L. Asbestos exposure and laryngeal cancer : an analysis of the epidemiologic evidence. Journal of occupational medicine. Vol.30, no.1(Jan. 1988). p.23-27.

Churg,A. Reply to Dr. Dunnigan. [Letter to the Editor]. American journal of industrial medicine. Vol.14(1988). p. 235-238.

Clarke,E.A.; Marrett,L.D.; Kreiger,N. Twenty years of cancer incidence (1964-1983): the Ontario Cancer Registry. Toronto: The Ontario Cancer Treatment and Research Foundation, 1987.

Craighead,J.M. Response to Dr. Dunnigan's commentary. [Letter to the Editor]. American journal of industrial medicine. Vol.14, no2(1988). p.241-243.

Damber,L.A.; Larsson,L.G. Occupation and male lung cancer: a case-control study in northern Sweden. British journal of industrial medicine. Vol.44(1987). p.446-453.

Davies,C.N. Diseases associated with the inhalation of asbestos dust. Arhiv za higijenu rada i toksikologiju. Vol.37(1986). p.253-274.

Davis,J.M. On the statement by Jacques Dunnigan. American journal of industrial medicine. Vol.14, no.5(1988). p.629-630.

Dement,J.M.; Harris.R.L.; et al. Exposures and mortality among chrysotile asbestos workers. PartII: Mortality. American journal of industrial medicine. Vol.4(1983). p.421-433.

Doll,R.; Peto,J. Effect on health of exposure to asbestos. Health and Safety Commission. London : Her Majesty's Stationery Office, 1985.

Dunnigam,J. Linking chrysotile asbestos with mesothelioma. American journal of industrial medicine. Vol.14,no.2(1988). p.205-209.

Edelman,D.A. Exposure to asbestos and the risk of gastrointestinal cancer: a reassessment. British journal of industrial medicine. Vol.45,no.2(Feb. 1988). p.75-82.

Elgie,R.G. [Referral of 87 03 05 asking the Panel to review and advise on recommendations from the Royal Commission on Asbestos regarding asbestos-related diseases].

Elmes,P.C.; Simpson,M.J.C. Insulation workers in Belfast. A further study of mortality due to asbestos exposure(1940-75). British journal of industrial medicine. Vol.34(1977). p.174-180.

Elwood,J.M.; Pearson,J.C.G.; et al. Alcohol, smoking, social and occupational factors in the aetiology of cancer of the oral cavity, pharynx and larynx. International journal of cancer. Vol.34,(1984). p.603-612.

Enterline,P.E.; Hartley,J.; Henderson,V. Asbestos and cancer: a cohort followed up to death. British journal of industrial medicine. Vol.44,no.6(1987). p. 396-401.

Enterline,P.E. Epidemiologic basis for the asbestos standard. Environmental health perspectives. Vol.52(1983). p.93-97.

Enterline,P.E. Cancer produced by nonoccupational asbestos exposure in the United States. Air Pollution Control Association Journal. Vol.33(1983). p.318-322.

Finkelstein,M.M. Mortality among employees of the Bendix Automotive Corporation, Windsor, Ontario. Toronto : Ontario Ministry of Labour, 1988.

Finkelstein,M.M. Mortality among former employees of the Domtar Pipe Factory in Cornwall, Ontario. Toronto : Ontario Ministry of Labour, 1987.

Finkelstein,M.M. Mortality among employees of a Sarnia, Ontario factory which manufactured insulation materials from amosite asbestos: a report and proposal for further study. Toronto: Ontario Ministry of Labour, 1987.

Finkelstein,M.M. Cancer among former employees of the Johns-Manville asbestos-cement factory in West Hill, Ontario: an updated analysis and assessment of risk. Toronto : Ontario Ministry of Labour,1988.

Finkelstein,M. On the relative toxicity of asbestos fibres. [Letter to the Editor]. British journal of industrial medicine. Vol.42(1985). p.69-70.

Finkelstein,M. Pulmonary function in asbestos cement workers : a dose-response study. British journal of industrial medicine. Vol.42(1986). p.406-413.

Finkelstein,M. A study of dose-response relationships for asbestos associated disease. British journal of industrial medicine. Vol.42,no.5. p.319-325.

Finkelstein,M. Mortality among employees of an Ontario asbestos-cement factory. American review of respiratory diseases. Vol.129(1984). p.754-761.

Fitzgerald,E.K. Asbestosis and neoplastic risk- conjugal contacts of asbestos workers- Scarborough,Ontario. Scarborough,Ont.: Department of Health, [1975].

Frumkin,H.; Berlin,J. Asbestos exposure and gastrointestinal malignancy review and meta-analysis. American journal of industrial medicine. Vol.14(1988). p.79-95.

Gardner,M.J.; Powell,C.A.; et al. Continuing high lung cancer mortality among ex-amosite asbestos factory workers and a pilot study of individual anti-smoking advice. Journal of the Society for Occupational Medicine. Vol.38(1988). p.69-72.

Gardner,M.J.; Acheson,E.D.; Winter,P.D. Mortality from mesothelioma of the pleura during 1968-78 in England and Wales. British journal of cancer. Vol.46(1982). p.81-88.

Gardner,M.J. Tumour incidence after asbestos exposure in Great Britain - with special reference to the cancer risk of the non-occupational population. VDI-Berichte. Vol.475(1983). p.185-190.

Garrahan,K. Mesothelioma : has patient had contact with even small amount of asbestos? Journal of the American Medical Association. Vol.257, no.12(1987). p.1569-70.

Gillette,J.; Weisburger,E.K. Strategies for determining the mechanisms of toxicity. Clinical toxicology. Vol.23,no.1(1985). p.1-78.

Goldsmith,J.R. Comparative epidemiology of men exposed to asbestos and man-made mineral fibers. American journal of industrial medicine. Vol.10(1986). p.543-552.

Goldsmith,J.R. Asbestos as a systemic carcinogen: the evidence from eleven cohorts. American journal of industrial medicine. Vol.3(1982). p.341-348.

Gough,M. Sources and interpretation of asbestos exposure data. Journal of toxicity and clinical toxicology. Vol.21, nos.1&2(1983-84). p.211-235.

Graham,S. Methodological problems in ecologic studies in the asbestos-cancer relationship. Environmental research. Vol.25(1981). p.35-49.

Greenberg,M.; Davies,T.A.L. Mesothelioma register 1967-68. British journal of industrial medicine. Vol.31(1974). p.91-104.

Hammond,E.C.; Garfinkel,L.; et al. Mortality experience of residents in the neighbourhood of an asbestos factory. Annals of the New York Academy of Science. Vol.330(1979). p.417-422.

Hammond,E.C.; Selikoff,I.J.; Seidman,H. Asbestos exposure, cigarette smoking, and death rates. Annals of the New York Academy of Sciences. Vol.330(1979). p.473-490.

Heller,R.M.; Janower,M.L.; Weber,A.L. The radiological manifestations of malignant pleural mesothelioma. American journal of roentgenology. Vol.108,no.1(1970). p.53-59.

Heller,J.G.; Martin,N.M. Surveillance and monitoring of asbestos workers by the Ministry of Labour. [Prepared for the Industrial Disease Standards Panel]. March 1, 1988.

Hillerdal,G. Malignant mesothelioma 1982 : review of 4710 published cases. British journal of diseases of the chest. Vol.77(1983). p.321-343.

Hinds,W.M.; Thomas,D.B.; O'Reilly,H.P. Asbestos, dental x-rays, tobacco, and alcohol in the epidemiology of laryngeal cancer. Cancer. Vol.44(1979). p.1114-1120.

Ho,S.F.; Lee,H.P.; Phoon,W.H. Malignant mesothelioma in Singapore. British journal of industrial medicine. Vol.44(1987). p.788-789.

Holstein,E.C.; Selikoff,I.J. Health status of workers 7-15 years after first employment in an asbestos insulation factory, with measured exposure; results of a survey in 1979. Report to the National Institute of Environmental Health Sciences. New York: Environmental Sciences Laboratory, Mount Sinai School of Medicine, City University of New York, 1980.

Hughes,J.M.; Weill,H.; Hammad,Y.Y. Mortality of workers employed in two asbestos cement manufacturing plants. American journal of industrial medicine. Vol.14(1988). p.639-657.

Hughes,J.M.; Weill,H. Asbestos exposure - quantitative assessment of risk. American review of respiratory disease. Vol.133(1986). p.5-13.

Huncharek,M. The biomedical and epidemiological characteristics of asbestos-related diseases : a review. Yale journal of biology and medicine. Vol.59(1986). p.435-451.

Huncharek,M. Asbestos-related mesothelioma risk among railroad workers. [Letter to the Editor]. American review of respiratory diseases. Vol.135(1987). p.983-984.

Industrial Disease Standards Panel. First report to the Workers' Compensation Board on certain issues arising from the Report of the Royal Commission on Asbestos. (IDSP report no.4). Toronto: Industrial Disease Standards Panel, September 1988.

Industrial Disease Standards Panel. First report on certain issues arising from the report of The Royal Commission on Asbestos. The Ontario Gazette. Vol.121-47(Nov.19,1988).

Injured Workers' Consultants. Response to Peter Barth's Workers' Compensation and Asbestos in Ontario. April, 1982.

Ison,T.G. Workers' compensation and asbestos in Ontario. A commentary on the report of Peter S. Barth. Both prepared for the Royal Commission on Matters of Health and Safety arising from the use of asbestos in Ontario. April, 1982.

Johnson,W.G.; Heler,E. The costs of asbestos-related disease and death. Milbank Memorial Fund Quarterly/Health and Society. Vol.61,no.2(1983). p.177-194.

Jones,J.S.P.; Smith,P.G.; et al. The consequences of exposure to asbestos dust in a wartime gas-mask factory. In : Wagner,J.C.; Smith, P.G., eds. Biological effects of mineral fibres. (IARC scientific publication no. 30). Lyon : International Agency for Research on Cancer, 1979. p.788-789.

Kaegi,E. [Response to letter of 1987 10 09 requesting information on the Board's policy on survivors' benefits for asbestos-related diseases, guidelines concerning the 'benefit of doubt' policy, and data on mesothelioma claims].

Kelsey,K.T. Approach to asbestos worker compensation plan alleged to be flawed. [Letter to the Editor]. Journal of occupational medicine. Vol.27(1985). p.790.

Kilburn,K.H.; Warshaw,R.; Thornton,J.C. Asbestos diseases and pulmonary symptoms and signs in shipyard workers and their families in Los Angeles. Archives of internal medicine. Vol.146(Nov.1986). p.2213-2220.

Kilburn,K.H.; Lilis,R.; et al. Asbestos disease in family contacts of shipyard workers. American journal of public health. Vol.75,no.6(June 1985). p.615-617.

Kjuus,H.; Skjaerven,R.; et al. A case-referent study of lung cancer, occupational exposures and smoking. II. Role of asbestos exposure. Scandinavian journal of work, environment and health. Vol.12(1986). p.203-209.

Knishkowy,B.; Baker,E.L. Transmission of occupational disease to family contacts. American journal of industrial medicine. Vol.9,no.6(1986). p.543-550.

Kogan,F.M. Re: chrysotile asbestos and mesothelioma of pleura. [Letter to the Editor]. American journal of industrial medicine. Vol.15, no.1(1989). p.123.

L'Abbe,K.A. Re: Statistics on asbestos related gastrointestinal cancers. March 12, 1990. [Internal memorandum].

Levine,D.S. Does asbestos exposure cause gastrointestinal cancer? Digestive diseases and sciences. Vol.30,no.12(December 1985). p.1189-1198.

Lewinsohn,H.C.; Meigs,J.W.; et al. The influence of occupational and environmental asbestos exposure on the incidence of malignant mesothelioma in Connecticut. (IARC scientific publication no.30). Lyon : International Agency for Research on Cancer, 1980. p.655-660.

Li,F.P.; Lokich,J.; et al. Familial mesothelioma after intense exposure at home. Journal of the American Medical Association. Vol.240,no.5(Aug.1978). p.467.

Liddell,F.D.K. Tumour incidence after asbestos exposure in the general population of Canada. VDI-Berichte. Vol.475(1983). p.179-183.

Liddell,F.D.K.; Hanley,J.A. Relations between asbestos exposure and lung cancer SMRs in occupational cohort studies. British journal of industrial medicine. Vol.42(1985). p.389-396.

Lieben,J. Mesothelioma and asbestos exposure. Archives of environmental health. Vol.14(April 1967). p.559-563.

Lilienfeld,D.E. Obfuscation : a reply to Dr. Dunnigan. American journal of industrial medicine. Vol.14,no.5(1988). p.625-628.

Lilienfeld,D.E.; Mandel,J.S. Projection of asbestos related diseases in the United States, 1985-2009 I. Cancer. British journal of industrial medicine. Vol.45(1988). p.283-291.

Lilis,R.; Selikoff,I.J.; et al. Asbestosis : interstitial pulmonary fibrosis and pleural fibrosis in a cohort of asbestos insulation workers: influence of cigarette smoking. American journal of industrial medicine. Vol.10(1986). p.459-470.

Lilis,R.; Selikoff,I.J.; et al. Asbestosis : interstitial pulmonary fibrosis and pleural fibrosis in a cohort of asbestos insulation workers: influence of cigarette smoking. American journal of industrial medicine. Vol.10(1986). p.459-470.

Lillington,G.A.; Jamplis,R.W.; et al. Conjugal malignant mesothelioma. The New England journal of medicine. Vol.291,no.11(Sept. 1974). p.583-584.

Luce,S.; Swimmer,G. Worker attitudes about health and safety in three asbestos brake manufacturing plants. [Report prepared for the Royal Commission on Matters of Health and Safety Arising from the use of Asbestos in Ontario]. (Study no.6). February, 1982.

Lynch,H.T.; Katz,D.; Markvicka,S.E. Familial mesothelioma : review and family study. Cancer genetics and cytogenetics. Vol.15,no.1-2(1985). p. 121-126.

Malker,H.S.R.; McLaughlin,J.K.; et al. Occupational risks for pleural mesothelioma in Sweden, 1961-79. Journal of the National Cancer Institute. Vol.74, no.1(Jan.1985). p.61-66.

Malker,H.S.; McLaughlin,J.K. Peritoneal mesothelioma in the construction industry in Sweden. Journal of occupational medicine. Vol.29, no.12(Dec.1987). p.979-980.

Mancuso,T.F. Relative risk of mesothelioma among railroad machinists exposed to chrysotile. American journal of industrial medicine. Vol.13(1988). p.639-657.

Mancuso,T.F. Mesothelioma among machinists in railroad and other industries. American journal of industrial medicine. Vol.4(1983). p.501-513.

Marsh,G.M. Critical review of epidemiologic studies related to ingested asbestos. Environmental health perspectives. Vol.53(1983). p.49-56.

Martensson,G.; Larsson,S.; Zettergren,L. Malignant mesothelioma in two pairs of siblings : is there a hereditary predisposing factor? European journal of respiratory diseases. Vol.65(1984). p.179-184.

McCracken,W.J. Comments and critique on a study prepared by Peter S. Barth, Workers' Compensation and Asbestos in Ontario, (January 1982) for the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. August 6, 1987.

McDonald,J.C. Cancer in chrysotile mines and mills. In: Biological effects of asbestos. (IARC scientific publication no.8). Lyon: International Agency for Research on Cancer, 1971. p.189-193.

McDonald,J.C. Asbestos-related disease : an epidemiological review. (IARC scientific publication no.30). Lyon : International Agency for Research on Cancer, 1980. p.587-601.

McDonald,J.C.; McDonald,A.D. Epidemiology of mesothelioma from estimated incidence. Preventive medicine. Vol.6(1977). p.426-446.

McDonald,A.D.; McDonald,J.C. Epidemiology of malignant mesothelioma. In: Asbestos-related malignancy. Orlando: Grune & Stratton, 1986. p.31-55.

McDonald,A.D.; Fry,J.S.; et al. Dust exposure and mortality in an American chrysotile textile plant. British journal of industrial medicine. Vol.40(1983). p.361-367.

McDonald,J.C. Tremolite, other amphiboles, and mesothelioma. [Letter to the Editor]. American journal of industrial medicine. Vol.14,no.2(1988). p.247-249.

McDonald,J.C. Health implications of environmental exposure to asbestos. Environmental health perspectives. Vol.62(1985). p.319-328.

McDonald,J.C. Silica silicosis, and lung cancer. [Editorial]. British journal of industrial medicine. Vol.46(1989). p.289-91.

McDonald,J.C.; McDonald,A.D.; et al. Cohort study of mortality of vermiculite miners exposed to tremolite. British journal of industrial medicine. Vol.43(1986). p.436-444.

McDonald,A.D.; Harper,A.; et al. Epidemiology of primary malignant mesothelial tumors in Canada. Cancer. Vol.26(Oct.1970). p.914-919.

McDonald,A.D.; Fry,J.S.; et al. Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. British journal of industrial medicine. Vol.39(1983). p.368-374.

McDonald,A.D. Mesothelioma registries in identifying asbestos hazards. Annals of the New York Academy of Sciences. Vol.330(1979). p.441-454.

McDonald,A.D.; McDonald,J.C. Mesothelioma after crocidolite exposure during gas mask manufacture. Environmental research. Vol.17(1978). p.340-346.

McDonald,J.C.; Liddell,F.D.K.; et al. Dust exposure and mortality in chrysotile mining, 1910-75. British journal of industrial medicine. Vol.37(1980). p.11-24.

Mcdonald,A.D.; McDonald,J.C. Malignant mesothelioma in North America. Cancer. Vol.46(1980). p.1650-1659.

McEwen,J.; Finlayson,A.; Mair,A. Asbestos and mesothelioma in Scotland. Internationales Archiv fur Arbeitsmedizin. Vol.28(1971). p.301-311.

Michaels,D. Asbestos disease in sheet metal workers: proportional mortality update. American journal of industrial medicine. Vol.13(1988). p.731-734.

Miller,A.B. Asbestos fibre dust and gastro-intestinal malignancies. Review of literature with regard to a cause/effect relationship. Journal of chronic diseases. Vol.31(1978). p.23-33.

Miller,A.B. Synthesis of papers on biological monitoring, case studies in screening, and social and economic issues. Journal of occupational medicine. Vol.28,no,8(Aug.1988). p.782-788.

Milne,J. Fifteen cases of pleural mesothelioma associated with occupational exposure to asbestos in Victoria. The medical journal of Australia. Vol.2(1969). p.669-670.

Monson,R.R. Occupational epidemiology. Boca Raton, Fla. : CRC Press, 1980.

Morgan,R.W.; Foliart,D.E.; Wong,O. Asbestos and gastrointestinal cancer. Western journal of medicine. Vol.143(July 1985). p.60-65.

Mowe,G.; Gylseth,B.; et al. Occupational asbestos exposure, lung fiber concentration and latency time in malignant mesothelioma. Scandinavian journal of work, environment and health. Vol.10(1984). p.293-298.

Mowe,G.; Gylseth,B. Medico-legal aspects of malignant mesothelioma. Scandinavian journal of society and medicine. Vol.12,no.1(1984). p.15-23.

Mowe,G.; Gylseth,B. Occupational asbestos exposure, lung-fiber concentration and latency time in malignant mesothelioma. Scandinavian journal of work, environment and health. Vol. 10(1984). p.293-298.

Newhouse,M.L.; Matthews,G.; et al. Mortality of workers at acetylene production plants. British journal of industrial medicine. Vol.45(1988). p.63-69.

Newhouse,M.L.; Berry,G.; Wagner,J.C. Mortality of factory workers in east London 1933-80. British journal of industrial medicine. Vol.42(1985). p.4-11.

Newhouse,M.L.; Thompson,H. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. British journal of industrial medicine. Vol.22(1965). p.261-269.

Newhouse,M.L.; Thompson,H. Epidemiology of mesothelial tumors in the London area. Annals of the New York Academy of Sciences. Vol.132(1965). p.579-588.

Nicholson,W.J. Case study 1: asbestos-- the TLV approach. Annals of the New York Academy of Sciences. Vol.271(1976). p.152-169.

Nicholson,W.J. Human cancer risk from ingested asbestos: a problem of uncertainty. Environmental health perspectives. Vol.53(1983). p.111-113.

Nicholson,W.J.; Selikoff,I.J.; et al. Long-term mortality experience of chrysotile miners and millers in Thetford Mines, Quebec. Annals of the New York Academy of Sciences. Vol.330(1979). p.11-21.

Nicholson,W.J. Tumour incidence after asbestos exposure in the USA: cancer risk of the non-occupational population. VDI-Berichte. Vol.475(1983). p.161-177.

Nicholson,W.J.; Rohl,A.N.; et al. Environmental asbestos concentrations in the United States. (IARC scientific publication no.2). Lyon: International Agency for Research on Cancer, 1980. p.823-827.

Nicholson,W.J. Airborne asbestos health assessment update. New York: Environmental and Occupational Medicine, Department of Community Medicine, Mount Sinai School of Medicine of the City University of New York, 1986.

Occupational Chest Disease Review Committee. Submission by the OCDRC to the Workers' Compensation Board in response to findings of the Industrial Disease Standards Panel. The Ontario Gazette. Vol.122-8(Feb.25,1989). p.763-780.

Ohlson,C.G.: Klaesson,B.: Hogstedt,C. Mortality among asbestos-exposed workers in a railroad workshop. Scandinavian journal of work, environment and health. Vol.10(1984). p.283-291.

Oliver,L.C.: Eisen,E.A.: et al. Asbestos-related disease in railroad workers. American review of respiratory diseases. Vol.131(1985). p.499-504.

Paci,E.: Dini,S.: et al. Malignant mesothelioma in non-asbestos textile workers in Florence. American journal of industrial medicine. Vol.11(1987). p.249-254.

Pampalon,R.: Siemiatycki,J.: Blanchet,M. Pollution environmentale par l'amiante et sante publique au Quebec. Union Medicale Canada. Vol.111(1982). p.475-489

Pelnar,P.V. Further evidence of non-asbestos-related mesothelioma. Scandinavian journal of work, environment, and health. Vol.14(1988). p.141-144.

Peterson,J.T.: Greenberg,S.D.: Buffler,P.A. Non-asbestos-related malignant mesothelioma : a review. Cancer. Vol.54(1984). p.951-960.

Peto,J. Problems in dose-response and risk assessment: the example of asbestos. Banbury report. Vol.19(1985). p.89-97.

Peto,J.: Doll,R.: et al. Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. Annals of occupational hygiene. Vol.29, no.3(1985). p.305-355.

Pinchin,D.J. Asbestos in buildings. [Report prepared for the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario]. (Study no.8). June, 1982.

Quinn,M.M.: Kriebel,D.: et al. An asbestos hazard in the reprocessed textile industry. American journal of industrial medicine. Vol.11(1987). p.255-266.

Raffn,E.: Lynge,E.: et al. Cancer incidence and mortality among employees at an asbestos-cement factory in Denmark. Copenhagen: Danish Cancer Registry,1988.

Rajhans,G.S.: Bragg,G.M.: Morton,J.S. A review of asbestos exposures in Ontario. American Industrial Hygiene Association journal. Vol.39(Sept.1978). p.767-771.

Richter,E.D.: Tulchinsky,T.: et al. Asbestos-exposed populations: prevention, care and compensation. Preventive medicine: Vol.13,no.3(1984). p.286-298.

Risberg,B.: Nickels,J.: Wagermark,J. Familial clustering of malignant mesothelioma. Cancer. Vol.45(1980). p.2422-2427.

Robinson,C.; Lemen,R.; Wagoner,J.K. Mortality patterns, 1940-1975 among workers employed in an asbestos textile friction and packing products manufacturing facility. In: Lemen,R.;Dement,R.,eds. Dusts and disease: proceedings of the conference on occupational exposures to fibrous and particulate dust and their extension into the environment. Forest Park, ILL.: Pathotox Publishers, 1979. p.131-143.

Roggli,V.L.; Pratt,P.C. Amphiboles and chrysotile asbestos exposure. [Letter to the Editor]. American journal of industrial medicine. Vol.14, no.2(1988). p.245-246.

Rossiter,C.E.; Coles,R.M. HM Dockyard,Devonport: 1947 mortality study. In: Wagner,J.C.;Davis,W.,eds. Biological effects of mineral fibres. (IARC scientific publication no.30). Lyon: International Agency for Research in Cancer, 1980. Vol.2. p.713-721.

Rubino,G.F.; Scansetti,G.; et al. Epidemiology of pleural mesothelioma in North-western Italy(Piedmont). British journal of industrial medicine. Vol.29(1972). p.436-442.

Rubino,G.F.; Piolatto,G.; et al. Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. British journal of industrial medicine. Vol.36(1979). p.187-194.

Rusby,N.L. Pleural manifestations following the inhalation of asbestos in relation to malignant change. Journal of the Royal Naval Medical Service. Vol.LIV,no.2(1968). p.436-442.

Sanden,A.; Jarvholm,B.; Naslund,P. Mortality and morbidity of Swedish insulation workers. [Letter to the Editor]. Scandinavian journal of work, environment and health. Vol.10(1984). p.207.

Schenker,M.B.; Garshick,E.; et al. A population-based case-control study of mesothelioma deaths among U.S. railroad workers. American review of respiratory diseases. Vol.134(1986). p.461-465.

Seidman,H.; Selikoff,I.J.; Gelb,S.K. Mortality experience of amosite asbestos factory workers : dose response relationships 5 to 40 years after onset of short-term work exposure. American journal of industrial medicine. Vol.10(1986). p. 479-514.

Selikoff,I.J.; Churg,J.; Hammond,E.C. The occurrence of asbestosis among insulation workers in the United States. Annals New York Academy of Sciences. Vol.132(Dec.1965). p.139-155.

Selikoff,I.J. Mortality experience of insulation workers in the United States and Canada, 1943-1976. Annals of the New York Academy of Sciences. Vol.330(1979). p.91-116.

Shalat,S.L.; True,L.D.; et al. Kidney cancer in utility workers exposed to polychlorinated biphenyls(PCBs). British journal of industrial medicine. Vol.46(1989). p.823-824.

Shannon,H.S.; Jamieson,E.; et al. Mortality experience of Ontario glass fibre workers - extended follow-up. Annals of occcupational hygiene. Vol.31,no.4B(1987). p.657-662.

Siemiatycki,J. Health effects on the general population (mortality in the general population in asbestos mining areas). In: Proceedings of the World Symposium on Asbestos. (1983). p.337-348.

Sluis-Cremer,G.K. Linking chrysotile asbestos with mesothelioma [letter to the editor]. American journal of industrial medicine. Vol.14,no.5(1988). p.631-632.

Smith,P.G.; Higgins,P.McR.; Park,W.D. Peritoneal mesothelioma presenting surgically. British journal of surgery. Vol.55(1968). p.681-684.

Solomons,K. Malignant mesothelioma - clinical and epidemiological features. South African medical journal. Vol.66(Sept.1984). p.407-412.

Stanbury,M.; Rosenman,K.D. A methodology for identifying workers exposed to asbestos since 1940. American journal of public health. Vol.77,no.7(July 1987). p.854-855.

Teta,M.J.; Lewinsohn,H.C.; et al. Mesothelioma in Connecticut, 1955-1977. Journal of occupational medicine. Vol.25,no.10(October 1983). p.749-756.

Theriault,G.P.; Grand-Bois,L. Mesothelioma and asbestos in the province of Quebec, 1969-1972. Archives of environmental health. Vol.3(1978). p.15-19.

Thomas,H.F.; Benjamin,I.T.; et al. Further follow-up study of workers from an asbestos cement factory. British journal of industrial medicine. Vol.39(1982). p.273-276.

Toogood,P. [Letter of 1988 03 16 re: mesothelioma incidence data for Ontario (including attached tabular information)].

Tuohy,C.J.; Trebilcock,M.J. Policy options in the regulation of asbestos-related health hazards. [Report prepared for the Royal Commission on Matters of Health and Safety Arising from the use of Asbestos in Ontario]. (Study no.3). January, 1982.

Verma,D.K.; Middleton,C.G. Occupational exposure to asbestos in the drywall taping process. American Industrial Hygiene Association journal. Vol.41(April 1980). p.264-269.

Vianna,N.J.; Polan,A.K. Non-occupational exposure to asbestos and malignant mesothelioma in females. The Lancet. (1978). p.1061-1063.

Wagner,J.C.; Sleggs,C.A.; Marchand,P. Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. British journal of industrial medicine. Vol.17(1960). p.260-271.

Wagner,J.C. Mesothelioma and mineral fibres. Cancer. Vol.57(May 15, 1986). p.1905-1911.

Wagner,J.C.; Newhouse,M.L.; et al. Correlation between fibre content of the lung and disease in east London asbestos factory workers. British journal of industrial medicine. Vol.45(1988). p.305-308.

Walker,A.M. Declining relative risks for lung cancer after cessation of asbestos exposure. Journal of occupational medicine. Vol.26,no.6(June 1984). p.422-426.

Walton,W.H. The nature, hazards and assessment of occupational exposure to airborne asbestos dust : a review. Annals of occupational hygiene. Vol.25,no.2(1982). p.117-247.

Weill,H.; Hughes,J.M. Asbestos as a public health risk : disease and policy. American review of public health. Vol.7(1986). p.171-192.

Weiss,W. Mortality of a cohort exposed to chrysotile asbestos. Journal of occupational medicine. Vol.19(Nov.1977). p.737-740.

Whitwell,F.; Scott,J.; Grimshaw,M. Relationship between occupations and asbestos-fibre content of the lungs in patients with pleural mesothelioma, lung cancer, and other diseases. Thorax. Vol.32(1977). p.377-386.

WHO; IARC; IACR. Cancer incidence in five continents. Vol.5. (IARC scientific publication no.88). Lyon: International Agency for Research on Cancer, 1987.

WHO. Asbestos and other natural mineral fibres. (WHO Environmental Health Criteria no. 53). Geneva: World Health Organization, [n.d.].

Wignall,B.K.; Fox,A.J. Mortality of female gas mask assemblers. British journal of industrial medicine. Vol.39(1982). p.34-38.

Woitowitz,H.-J.; Lange,H.-J.; et al. Mortality rates in the Federal Republic of Germany following previous occupational exposure to asbestos dust. International archives of occupational and environmental health. Vol.57(1986). p.161-171.

Workers' Compensation Board of Ontario. [Response of the Workers' Compensation Board to the recommendations contained in the Industrial Disease Standards Panel's report no.4 on certain issues arising from the Report of the Royal Commission on Asbestos]. Oct.21, 1989. The Ontario gazette. Vol.122-42 (Oct.21,1989). p.5741-5751.

Workers' Compensation Board. Comments on the study and review papers on Workmen's Compensation and Asbestos in Ontario. [Report filed with the Royal Commission on Matters of Health and Safety Arising from the use of Asbestos in Ontario]. August 6, 1982.

Wright,W.E.; Sherwin,R.P.; et al. Malignant mesothelioma: incidence, asbestos exposure, and reclassification of histopathology. British journal of industrial medicine. Vol.41(1984). p.39-45.

Yeung,K.S.; Heller,J.G. Report on the relationship between occupational asbestos exposure and malignant diseases. July 5, 1989. [Prepared for the IDSP].

Zoloth,S.; Michaels,D. Asbestos disease in sheet metal workers: the results of a proportional mortality analysis. American journal of industrial medicine. Vol.7(1985). p.315-321.